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EXPECTED LAGS BETWEEN CLINICAL DIAGNOSIS AND X-RAY CONFIRMATION*

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IN the great progress that has been made in the application of roentgenology to diagnosis, and none would fail to acknowledge this progress to have been a tremendous one, clinicians often lose sight of certain inevitable limitations to this valuable method of diagnosis. limitations inevitable by reason of the relationship between the x-ray and its penetration through different tissues in the body and through different substances that can be introduced into the body. As a result of this they may accept a negative x-ray report as contraindicating a diagnosis made on clinical grounds. With proper understanding of the matter this should not occur.

Until certain structural changes, which are demonstrable by x-ray, have occurred, there is nothing that can be demonstrated by x-ray. In some diseases such structural changes never occur; in others they occur only after well-marked symptoms have developed; in still others structural changes demonstrable by x-ray and symptoms appear almost simultaneously; not infrequently the changes demonstrable by x-ray antedate the development of definite symptoms. In the first group come diseases like typhoid fever, Bright's disease, cirrhosis of the liver, many diseases of the central nervous system, etc., in which x-ray gives no help in diagnosis. It is the second group to which at this time I will direct attention for reasons already indicated in earlier sentences of this paper, giving some examples of what I term expected lag between clinical and x-ray confirmation.

Severe, persisting, backache is a common condition. As probable causes we recognize, among others, osteoarthritis of the spine and neoplasm of the vertebrae. The aid of roentgenology is sought. Clinically there is strong suspicion that neoplasm is the causative factor. The roentgenologist reports no evidence of neoplasm involving the bone, or he may report evidences of osteoarthritis. Does such report justify the clinician in giving up his diagnosis of neoplasm? No, I have observed patients with such x-ray reports in whom the pathologist soon could demonstrate neoplasm or in whom later on the x-ray showed tumor; if arthritis was present, it was not a cause of pain. I will cite briefly such a patient.

A woman of fifty-two came to the hospital complaining of pain in the back of four months' duration, recently very severe. The right lobe of the thyroid was enlarged, irregular and very hard. The diagnosis of malignant tumor of thyroid with metastases in the spine was made, and this patient was demonstrated by me as such at a clinic held on November 22. The x-ray report on November 8 was as follows: "Anteroposterior films of thoracic and lumbar spine and pelvis show no evidence of metastases. There are only slight arthritic changes." X-ray, repeated on November 20, was reported as follows: "Anteroposterior stereo of the spine and lateral shows no definite evidence of metastasis. Vertebrae appear slightly decalcified, and there are changes in the articular processes of fourth and fifth thoracic, but these probably are

FIGURE 1. 1st, 2nd and 3rd lumbar vertebrae showing tumor metastases.

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arthritic." The patient died December 17, and at autopsy there was found carcinoma of the thyroid with metastases to regional tissues, mediastinum, liver, lungs, vertebrae, ribs, left iliac bone and intercostal muscles. What particularly interests us in this connection are the findings in the vertebrae not revealed by x-ray but obviously of a nature to be the cause of the pain in the back that the patient complained of. "On examining the vertebral column the second lumbar vertebra is seen to be involved by tumor growth. It is extremely soft and presents a reddish gray color instead of white. It is so soft that slightest pressure causes a compression of the vertebra. A saw cut made through the first, second and third lumbar vertebrae (fig. 1) shows that the entire anterior half of the second lumbar vertebra is completely destroyed by tumor tissue. The bone is extremely soft, reddish gray in color, almost fluid in consistency. The posterior half also is involved by tumor. Here the tissue is firmer and has a more whitish gray aspect. The first lumbar vertebra also shows a large tumor nodule approximately 1½ cm. in diameter, which is reddish gray in color and shows a central area of necrosis of approximately 1 cm. in diameter. Here the tissue is fluid in consistency. The third lumbar vertebra shows a similar area of metastasis, which is approximately 1½ cm. in diameter, the larger portion of which is necrotic and fluid in consistency."

It is well to remember in this connection that after the age of fifty, osteoarthritic changes in the spine are very frequent; they may be symptomless, even when very marked as shown by x-ray.

Pleural fluid, recurring several times following paracentesis, in a patient without evidences of cardiac insufficiency justifies a clinical diagnosis of neoplasm as the cause even when the roentgenologist reports no evidence of tumor. The interval between such clinical diagnosis and x-ray confirmation may be a long one as shown by the following history:

A man, aged 49, was admitted to the hospital on October 24, 1932 with the physical signs of right-sided pleural effusion. He had previously been in the Out-Door Department and on October 6, 1932 an x-ray showed the right diaphragm obscured by fluid which rose in the axilla; the right chest appeared flattened and the right lower border of the lung was atelectatic; fluoroscopy showed a collection of fluid in the septum between the upper and lower lobes on the right; the posterior mediastinum was clear.

On the day of admission to the hospital 1,400 cc. of fluid was removed from the right chest with a specific gravity of 1.018 and a cell count of 24 lymphocytes and 41 red blood cells. No bacteria could be found either microscopically or on culture. On November 3, 1,100 cc. fluid was removed from the same chest, on November 15, 1,050 cc. and on November 25, 700 cc.

He remained in the hospital until November 29, 1932. On November 26 the sixth x-ray examination of his chest gave the following: "Stereo x-ray still shows mottling of the right lower lobe but only a small amount of fluid. The process in the right upper posteriorly is unchanged. There is no essential change since previous examination." The previous examination contained the statement "there is no evidence of neoplasm".

During his stay in the hospital this patient was demonstrated by me in a clinic as an example of pleural effusion due to neoplasm though x-ray had failed to show any evidences of this.

He was readmitted to the hospital on February 28, 1933 and remained until March 4, 1933. During this stay his chest was tapped twice, once with the removal of 1,600 cc. and once with the removal of 400 cc. Careful study of various fluids showed no cells suspicious of tumor origin.

On March 3, 1933, a ninth roentgen examination of the chest was reported to show only slight further clearing of the right base. There was still enough fluid to obliterate the diaphragm and costophrenic angle. Fluoroscopy gave no additional information. Five months after the first x-ray showing fluid in the pleura there was still no x-ray evidence of tumor.

This patient was lost sight of until, on March 15, 1934, in a letter from Dr. E. D. Churchill of the Massachusetts General Hospital in regard to him the following statement is made: "Several weeks ago I explored this man's chest and found a primary carcinoma of the lung situated in the upper lobe with metastasis to the visceral pleura in the upper mediastinum. Microscopic section of a biopsy from this metastasis showed adenocarcinoma." This operation was almost a year and a half after pleural fluid had developed and nearly as long since the patient had been presented in a clinic as an example of pleural fluid due to tumor.

Prolonged fever often is difficult to explain. In the absence of demonstrable causes of various sorts, neoplasm should be considered as the cause and diligently sought for by all possible methods of examination including of course x-ray studies such as may be applied. Sometimes such x-ray studies are negative, when later they demonstrate a neoplasm; in other words, a neoplasm causing the symptoms of prolonged fever may fail to show in x-ray until further time has elapsed for the neoplasm to progress to x-ray visibility. Bone metastases may become demonstrable later and explain such a fever, just as has been indicated in the instance of back pain. Lung tumor similarly may become demonstrable as in the following patient:

A woman of sixty-four came to the hospital on February 3 with the story that for some four months there had been progressing fatigue, some cough, fever, a marked change in personality with depression and with recent confusion and disorientation. On December 22 she had been seen by a colleague, who could not make an adequate diagnosis; physical examination gave no definite findings. A week before he saw her, an x-ray examination of her chest, made on account of cough, had shown only slight peribronchial enlargement at the right hilus. When she came to the Peter Bent Brigham Hospital on February 3 the liver was considerably enlarged, and there were signs of fluid, small in amount, in each pleural sac. X-ray of the chest showed a small, rounded mass in the right hilus, 3.5 cm. in diameter, suggesting to the roentgenologist a metatasis rather than primary carcinoma. There was a small amount of fluid in each costophrenic angle, slightly more on the left. She died on February 14, and autopsy showed primary bronchiogenic carcinoma at hilum of right lung with extensive metastases. Neoplasm was the cause of the fever and other symptoms.

A patient's history may be strikingly indicative of pulmonary abscess following tonsillec-

tomy, extraction of teeth or pneumonia. There is fever; sputum may be abundant, sometimes foul; physical signs indicate a local pulmonary process; a clinical diagnosis of lung abscess is made; x-ray shows no evidences of abscess but does show a localized shadow from infiltration of the lung, a pneumonic consolidation; later a repeat x-ray shows a suggestion of abscess; the next x-ray shows an abscess cavity, probably with fluid level. The x-ray lag in such cases may be very long as in the following patient (42970), but it averages in my experience about ten days.

A Negro man, aged forty, was admitted to the hospital May 8, 1933 giving the following history. For approximately one month the patient has had a paroxysmal cough productive of large amounts of foul-smelling sputum. During this period he has had drenching night sweats. On the morning of admission during a paroxysm of coughing he had a sensation of something giving away in his left upper chest, and immediately thereafter he raised a large quantity of extremely foul-smelling sputum. There is no history of aspiration of foreign body or previous cold in the chest. Two years ago the patient had a tooth extracted, and he has had dental caries for years.

Physical examination of the lungs showed a normal right lung. On the left side behind from the mid-portion of the scapula half way down to the base there was moderate dullness in an ill-defined area. Over this area breath and voice sounds were slightly diminished, but breath sounds were harsh in character and at times suggestively tubular. There were numerous coarse râles on both inspiration and expiration. The rest of the left lung seemed normal. The clinical diagnosis of lung abscess was made.

On May 9 stereo x-ray of the chest showed the right lung to be clear. There was diffuse clouding through the middle third of the left lung with a slight increase in the markings extending toward the base. "The process is apparently in the posterior portion of the chest and is probably in the upper portion of the lower lobe. No definite cavity formation seen. Impression: pneumonic process in the left lower lobe."

Two weeks later, on May 23, reëxamination of the chest showed a definite spread of the process in the left lung which now involves the upper two-thirds, most marked around the hilus. "There are now several indistinct small clear areas posteriorly which may be beginning cavities. Heart and trachea are not displaced. Impression: pneumonic consolidation, probably beginning pulmonary abscess."

A third x-ray examination on June 10, a month after the first one, showed the extent of involvement slightly less than at previous examination with clearing of the lower portion. "There is now a definite cavity rather deep in the lung in line with the first interspace anteriorly, fifth rib posteriorly, 6 cm. from the midline. There is definite clouding of the pleura over the left upper lobe. Left diaphragm is elevated and slightly limited. Impression: pulmonary abscess."

Additional x-rays were taken and on July 14 the fifth x-ray examination of the chest showed marked clearing of the process in the left upper lobe, and the cavity had disappeared. "There is now only slight consolidation around the left hilus extending into the left upper lobe. Right lung remains clear."

It might be put thus, something in the lung that is not, namely a cavity, is not roentgenologically demonstrable, if the surrounding exudation in the pulmonary tissue in relation to the cavity is relatively extensive, or if the cavity is filled with exudate similar to that in the surrounding parenchyma. These are conditions which exist almost always in some stage of lung abscess and hence the delay in their demonstration by x-ray.

Bronchial stenosis often gives definitive physical signs when x-ray may show no positive evidences of change in the lung parenchyma. X-ray, obviously, may fail to show anything to indicate the obstructing lesion in the bronchus, if there is no peribronchial lesion at the point of the partial stenosis. Later on, when obstruction becomes more marked, x-ray will demonstrate the partial or complete airlessness of that portion of the lung.

Osteomyelitis is another condition in which there should be recognized a very considerable lag between symptoms and x-ray confirmation of diagnosis, whether we are dealing with long bones or flat bones. In mastoiditis, not realizing this may cause serious delay in performing the needed operation. Cause of symptoms indicating septicemia may be missed by reason of x-ray not showing osteomyelitis, let us say in the femur. The lag in osteomyelitis is variously stated as one to several weeks.

Arthritis is still another example. It is surprising often how little the x-ray can demonstrate in a very obvious disabling rheumatoid arthritis after a long duration of symptoms. Gouty changes in joints notably may give no evidence of gout when the joints are studied roentgenographically. The roentgenologist is not justified in saying, as he sometimes does, by x-ray this condition is chronic osteoarthritis, not gout. Often one sees deposits of uratic material on the cartilaginous surfaces of joints which in no way could be evident in the x-ray film.

I have proved a pulmonary tuberculosis, suspected from history including recent hemoptysis and from the hearing of a few localized râles, by finding tubercle bacilli at a time when the x-ray showed no lesion; later x-ray revealed a parenchymal lesion in the region where râles had been heard. Of course, far more frequent is it for the x-ray to reveal pulmonary tuberculosis, only suspected from history, of whose occurrence no other evidence could be found.

Also there may be a lag between symptoms and x-ray demonstration of cancer of the stomach or bowel. Sometimes the clinical story is so suggestive as to point definitely to the diagnosis and yet x-ray is not confirmatory; much more often the clinical picture is only definite enough to suggest some disturbance of stomach or bowel. I have seen some definite examples of failure of the x-ray to show the lesion in stomach or bowel in such patients when repeat x-rays a few weeks later showed unmistakable

evidence of cancer; this has occurred oftenest with cancer of the stomach, less frequently with cancer of the intestine.

Very probably other examples might be offered, but I am not attempting a full category of conditions in which a lag between clinical diagnosis and x-ray confirmation can occur. I am merely pointing out that it not infrequently does occur.

In the above I have sought to present by a few examples a phase of the problem of x-ray in diagnosis, all too often not sufficiently realized by clinicians and not appreciated by poorly trained roentgenologists. For him who understands the fundamental principles of x-ray in diagnosis, and this of course includes all well-trained roentgenologists, it is not necessary to point out that in some conditions a lag between clinical diagnosis and x-ray confirmation is to be expected, a lag which lengthens with the increased experience and diagnostic acumen of the clinician, and which shortens with the increased experience and skill of the roentgenologist and with improvement in the apparatus with which he works. As clinicians, we should not demand the impossible of roentgenology. The possible, however, is of the very greatest help in diagnosis, and this clinicians should acknowledge.

From the above discussion the superficial reader might draw the conclusion that the author is belittling the value of the x-ray in diagnosis. This is not the case; both positive and negative x-ray findings are of the very greatest value in diagnosis in confirming or ruling out suspected lesions as well as in not infrequently revealing lesions totally unsuspected from history and physical examination. However, it needs to be recognized that there are definite limitations to what x-ray methods can show, and these limitations should be better recognized by both clinicians and roentgenologists.

ELECTROSURGICAL CHOLECYSTECTOMY*

I. Experimental Observations

BY LESTER R. WHITAKER, M.D.†

HEMORRHAGE is the primary consideration and the chief danger in section of the tissues. Before the days of anesthesia attention was paid only to gross bleeding which was controlled by the tourniquet or mass ligature. The surgeon had no time to spend clamping and tying small vessels. He worked with utmost speed in the effort to finish his operation before the patient died of pain, hemorrhage, and shock. The old tradition of haste still exerts a surprising influence on surgical technique. Even though anesthesia gave the operator a quiet patient and time in which to do careful anatomical dissection, individual ligation of vessels, and other refinements, many to this day rush through even a simple operation as if they expected the patient to die before they could finish. Among surgeons of past generations there was a dictum that no major abdominal operation should consume more than one hour of time; if it approached two hours, there was danger of losing the patient. These surgeons acted accordingly. They made large incisions, handled the tissues hurriedly and roughly, exposed viscera to chilling and drying, and ligated only the vessels which spurted in their faces. After an hour of this of course the patient was almost invariably in a condition of shock.

Gradually a school was developed, of which Halsted was a leader, where men began thinking for themselves with regard to the adaptation of treatment to conditions, rather than learning technique in an apprenticeship manner. These men took advantage of the extra time allowed by anesthesia to handle and pack away viscera carefully, make anatomical dissections, ligate individual bleeders; and in closure of the wound to obtain apposition of tissues. These methods had the advantages of producing the least trauma and disturbance of the anatomy; and allowing less opportunity for collection of blood and serum in the wound, thus favoring primary healing and rapid recovery of the patient. Any surgical operation is an insult to the human frame; it should be made as light as possible.

The great advantage of electrosurgery is in control of hemorrhage. The cutting current (acusection) seals the capillaries and small vessels, preventing oozing, and allowing the "spurters" to be more easily and accurately clamped. Medium-sized vessels can be snapped and the tissue in the jaws of the clamp coagulated by holding in contact with the active electrode. Larger vessels, however, should not be treated in this way because the coagulum loosens after a day or two and there is risk of secondary hemorrhage. Tissue to be removed, such as tumors, may be handled either by direct attack with the cutting current, using a loop electrode; or after electrocoagulation en masse. The former can be used only in case the tissue is not too vascular. *In very vascular tumors, or organs such as the liver, kidney, and spleen, masses of tissue must be treated first with the coagulating cur-*

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rent by insertion of a needle electrode at intervals, reducing the tissue to a firm coagulum which can then be removed by the cutting current without hemorrhage[1].

Pribram in 1928[2], published a method of treating the acutely inflamed, gangrenous, or sclerotic gall-bladder with the cautery to dispose of it and at the same time reduce the danger of hemorrhage. By sterilizing the infected mucous membrane and tissues of the gall-bladder with the actual cautery Pribram felt that he had avoided the necessity for drainage. The description of Pribram's method follows:

"In order to avoid injury to the liver-tissue in cases where subserous resection (shelling out) was not successful, I have tried another way in order to be able to close the abdominal cavity. The vesicle is completely emptied of its liquid contents by the water pump; then split through the middle with the scissors from the fundus to the cystic duct; and the stones removed. The exposed cystic duct is cut between two ligatures. Then the mucous membrane is completely charred with a cautery down to the serosa. In this way any abscess of the wall penetrating the liver is completely destroyed. Then the two edges of the gall-bladder are sewn exactly together. To this procedure I have given the name '*mukoklase*' (destruction of mucosa). It has proved entirely satisfactory in all the cases where I have used it and is the method of choice for making a complete wound closure in complicated cases."

Pribram recommends the procedure in cases of shrunken gall-bladder where resection is difficult because of sclerosis; also in badly infected and gangrenous stone-containing vesicles, and in empyema. He has had only three deaths in two hundred cases, a remarkable record. He attributes the rapid convalescence and low mortality partially to the absence of drainage, avoiding, as he believes, peritonitis, acute dilatation of the stomach, erosion of blood vessels with secondary hemorrhage, postoperative pulmonary and cardiac complications.

The first impression from reading a description of Pribram's method was that the use of an actual cautery deep in the abdominal cavity was rather heroic, especially in close proximity to such vital structures as the bile-duct and associated vessels. The second thought was that a great deal of dead tissue was left to be absorbed. And thirdly, it seemed rather doubtful that this could be done safely without drainage. However, in favor of the method were Pribram's remarkable results; statistics which compared favorably even with those where only a slightly diseased gall-bladder had been removed by the usual dissection method.

EXPERIMENTS WITH OBLITERATION OF THE GALL-BLADDER BY DIFFERENT MEANS

Where possible of adaptation every radically new technical procedure in abdominal surgery should be tried first upon the dog. The originator of a method may be very skillful in its use but an imitator naturally cannot be until he has had considerable practice. Let this risk be taken by the dog. For several years I have been concerned with methods for removing or obliterating the gall-bladder with the greatest safety and the least possible disturbance to the patient. William J. Mayo, in 1900, presented a method for removal of the mucous membrane of the gall-bladder as a substitute for cholecystectomy[3]. He considered that if the mucous membrane were removed the gall-bladder would become obliterated. I tried the removal of the mucous membrane in gall-bladders of dogs but found it extremely difficult to make the dissection, and came to the conclusion that cholecystectomy would be more easily performed, particularly the subserous removal described by Doyen[4]. By using a headlight and special retractors a gall-bladder not too sclerotic can be removed subserously through a very small incision, allowing the patient to make an extremely rapid recovery[5, 6]. With one patient having gall-stones who was a poor risk for cholecystectomy a method of destroying the mucous membrane of the gall-bladder was tried by chemical cauterization. Although several attempts were made to obliterate the sinus with formalin, carbolic acid, and alcohol, the mucous membrane would regenerate each time and the opening remained until the patient died of cancer of the rectum about a year later. The difficulty with all operations upon the mucous membrane is the achievement of total destruction. Experiments upon dogs have shown that even a small section left near the cystic duct will regenerate.

AN ATTEMPT TO OBLITERATE THE GALL-BLADDER BY CAUTERIZATION OF THE CYSTIC DUCT

The publication of Pribram's method renewed my interest in these safer and less traumatic methods for removal of the gall-bladder. It seemed possible to go a step farther than Pribram. By his method, considerable tissue of the gall-bladder had to be sloughed and absorbed, yet good results were obtained. The lymphatics and veins of the gall-bladder are in the wall of the cystic duct. Why could not one by destroying this vital part of the gall-bladder produce necrosis of the vesicle, allowing it to slough away through a drainage tract; thus accomplishing its removal in patients who were poor risks by an easy technical procedure with little trauma? A piece of iron was fashioned to fit the outlet of the cystic duct in a dog. Then with a small abdominal incision and the gall-

bladder opened at the fundus the iron was heated, not red hot, but enough to produce some searing of the tissue. When applied inside the cystic duct it had a tendency to stick to the mucous membrane, requiring considerable force to tear it away. After cauterization a soft rubber tube was placed in the gall-bladder, taking care not to press it down into the duct enough to produce necrosis of the wall. This method was tried in three dogs. One of them, sacrificed after one day, showed the gall-bladder collapsed around the drainage tube, edematous and inflamed, with a slight injection of the surrounding peritoneum. Another dog, sacrificed after eight days, had peritonitis, the gall-bladder was necrotic, and cystic duct leaking. The third dog made only a fair postoperative recovery for three days but was lost, no autopsy being performed. These poor results led us to discard the method of destruction of the gall-bladder by cauterization of the cystic duct.

AN ATTEMPT TO OBLITERATE THE GALL-BLADDER BY CAUTERIZATION OF THE MUCOSA

With ten dogs the whole mucosa of the gall-bladder was cauterized to varying degrees, a soft drainage tube being placed inside. In four, light cauterization was done. These made good immediate postoperative recoveries. One dog, sacrificed after one day, showed a gall-bladder collapsed about the tube and inflamed, with adhesions to the omentum. Two, sacrificed after six and eight days, showed no peritonitis, but adhesions of omentum to a sloughing gall-bladder; with one of them, however, the slough was small, and it was considered that the gall-bladder would have been obliterated. Another dog, sacrificed after eleven days, showed peritonitis with omental adhesions around a necrotic gall-bladder.

Out of the six dogs in which moderate cauterization was done, three had peritonitis, one with a hole from the gall-bladder into the peritoneal cavity. The three other dogs, sacrificed after one to twelve days, had necrotic gall-bladders covered by omental adhesions, one with damage to the liver.

In two additional dogs the gall-bladder was damaged by clamping eleven days previous to the cauterization; the gall-bladder was rendered slightly thickened and sclerotic thereby. In these two the whole mucosa of the gall-bladder was cauterized by the hot iron and a soft drainage tube was placed inside. Both dogs made good immediate postoperative recoveries; one showed slight damage to the liver about the gall-bladder, with adhesions; the other, no adhesions; neither had peritonitis, and the ducts were normal. Apparently the actual cautery worked better in the slightly sclerotic gall-bladder.

In two cats the mucosa of the gall-bladder was cauterized and collapsed by stitching as with the Pribram method. Autopsy after eight days showed marked peritonitis with leaking, necrotic gall-bladders in both. A cat which had a tube placed in the gall-bladder (removed the third day) showed, after eight days, an excellent result—apparent obliteration of the gall-bladder.

A summary of results with use of the actual cautery on the mucosa of the gall-bladder in twelve dogs and three cats reveals good recoveries, after six to eight days, in six dogs and one cat. There was liver damage in two dogs; peritonitis, after eight to twelve days, in three dogs and two cats; necrotic gall-bladders, after eight to twelve days, in six dogs and two cats. In general, light cauterization of the mucosa gave better results than heavy, but necrosis resulted even with that. Heavy cauterization produced liver damage. The difficulty with a hot iron is in limiting its action to the mucosa without causing too rapid necrosis of the gall-bladder or damage to the liver. The best results were obtained with gall-bladders rendered slightly sclerotic by previous mechanical damage.

THE USE OF THE HIGH FREQUENCY ELECTRIC CURRENT FOR OBLITERATION OF THE GALL-BLADDER

It is obvious from the foregoing experiments that the difficulty with the actual cautery is to control the degree of destruction of the tissues. The best results were obtained with light cauterization. The high frequency current from the electrosurgical unit allows much better adaptation. For these experiments the Bovie Unit, old style, was used.

The tissue dissolving current, the "cutting current", was employed with six dogs. The ball electrode with the power control set from fifteen to twenty-five, low voltage and light dehydration, was run around over the mucosa of the gall-bladder, a drainage tube then being placed inside. Generally too much tissue destruction was produced. Good results were obtained in three of the dogs, those in which light treatment to the mucosa was given. The other three had necrosis of the gall-bladder, and two of these, peritonitis.

In two dogs the gall-bladder was damaged previously by clamping over the surface, which resulted in considerable inflammatory thickening with adherent omentum. In both cases the mucosa was treated moderately with the cutting current. The gall-bladder was first split to the cystic duct which was tied with silk. The ball electrode was run over the mucosa and the gall-bladder collapsed with a continuous stitch, no drainage being employed. Autopsy after several weeks showed a good result in obliteration of the gall-bladder in one of the dogs; in the other it was only partly obliterated, having reformed somewhat from the cystic-duct end. This tissue-dissolving current, as well as the actual cautery, apparently worked better with thickened, inflammatory gall-bladders, but likewise

it seemed to be too difficult of control for safe use.

TREATMENT OF THE MUCOSA BY THE COAGULATING CURRENT

The method of Pribram was used with seven dogs, simply substituting the coagulating current (biterminal) from the electrosurgical unit for the actual cautery. The gall-bladder was split to the cystic duct by using the cutting current, with heavy dehydration to help control hemorrhage. The spurting bleeders were snapped in the edge of the cut wall of the gall-bladder and tied. The cystic duct was dissected free and tied. The mucosa was treated by fulguration (sparking) making occasional contact of the needle with the tissue. Treatment was rather thorough to prevent regeneration. Then according to Pribram's method, the leaves of the gall-bladder were sewn together and the cavity obliterated. No drainage was used.

Three of the seven dogs made good postoperative recoveries. One of them, sacrificed after four days, showed only slight sloughing of the gall-bladder which probably would have been resolved. Another, sacrificed after two weeks, showed an excellent result, the remains of the gall-bladder being fibrous and covered with omental adhesions. This is the ideal, and the best that can be expected after leaving so much dead tissue in place. The third dog was in excellent condition after two weeks.

In the other four of the seven dogs poor results were obtained. One of them died of peritonitis five days after the operation; three died of intraperitoneal hemorrhage, after one, two, and eight days respectively; two of them showed a sloughing gall-bladder.

With two additional dogs the gall-bladder was damaged by clamping nine days before the Pribram method was applied. Good results were obtained in both these animals, sacrificed after ten and eleven weeks. In one, however, the gall-bladder had regenerated to about one-half its normal size, again emphasizing the necessity for complete destruction of the mucosa with this type of operation. In the other dog there were numerous adhesions of the omentum and adjacent viscera to the gall-bladder fossa, this being the expected result.

It is to be noted that the use of fulguration upon the non-sclerotic gall-bladder is liable to result in hemorrhage. The probability is that the vessels are so little supported that they bleed when the charred tissue is dissolved. It will be remembered that with the sclerotic gall-bladder the cutting or tissue-dissolving current was safer and more effective. The same is observed with the use of fulguration from the coagulating current.

In two dogs the uniterminal coagulating current (desiccation) was applied through the flat blade electrode which was rubbed over the mucosa rather lightly, the gall-bladder being sutured according to the regular Pribram procedure. Both these dogs died; one the same day, and the other after six days. Both showed hemorrhage into the peritoneal cavity; one of them bile leakage. Probably with the uniterminal current the tissue destruction is too great, weakening the walls of large vessels; or the depth of coagulation is not sufficient to seal off the vessels for a period long enough to allow a firm clot to form in them before the ends open.

MODIFIED PRIBRAM OPERATION, USING COAGULATING CURRENT (BITERMINAL)

Following these experiments, while observing a cholecystectomy at the Massachusetts Memorial Hospitals upon a chronically thickened and subacutely inflamed gall-bladder, I saw the surgeon, W. K. S. Thomas, split the vesicle to the cystic duct along the front with scissors, and then trim off the two leaves a short distance from their attachment to the liver, clamping and tying the bleeders encountered. This suggested a modification of the Pribram operation by removing the redundant parts of the gall-bladder for the purpose of reducing the amount of dead tissue to be disposed of by autolysis.

In four dogs a combination of this method and that of Pribram, using the electrosurgical unit, was tried. The gall-bladder was split to the cystic duct using the cutting current, heavy dehydration; then the leaves of the gall-bladder were cut away about one centimeter from the attachment to the liver, clamping and tying the bleeding vessels. The cystic duct was dissected free and tied. The mucosa of the part of the gall-bladder left attached to the liver was thoroughly fulgurated with the coagulating current[7]. In three of the four dogs the edges of this coagulated tissue were whipped over with a continuous stitch; *no drainage* was employed.

Two of the four dogs made good recoveries, one of these being the subject in which the gall-bladder edges were not stitched after the coagulation. These two dogs were sacrificed after two months. In one of them the gall-bladder had reformed to the size of a walnut, again emphasizing the necessity for tying the cystic duct low down, and of being sure to kill all the mucosa of the gall-bladder with the coagulating current. The dog in which the remainder of the gall-bladder was not stitched after the coagulation showed an excellent result. The stomach at the pylorus, however, was fixed to the gall-bladder fossa with pronounced adhesions. The two other dogs died the first day after the operation, from hemorrhage into the abdominal cavity; one of them showed a large clot of blood in the gall-bladder fossa. Probably with some of the larger vessels the coagulum dissolved rapidly and allowed bleeding. This suggests that larger vessels should be tied; and the tissue

about them should not be treated with fulguration.

In three additional dogs the gall-bladder had been damaged previously; in one of them, nine days before the cholecystectomy, by clamping; and in the others, three months before the operation, by the application of ninety-five per cent phenol. In the first, the gall-bladder was acutely inflamed, and in the other two slightly sclerotic. At the operation the gall-bladder was split to the cystic duct with the cutting current, using heavy dehydration; the bleeders were clamped and tied. Then the redundant portions of the gall-bladder were cut away with the same current about a centimeter from the attachment to the liver, the bleeders again being clamped and tied. With the coagulating current, using *both fulguration and contact coagulation*, for thoroughness, the whole surface of the portion of the gall-bladder left attached to the liver was treated, *taking care to avoid the points where vessels were tied.* The dogs were sacrificed from two to six months after the operation, having been in perfect health and showing at autopsy only a few adhesions of omentum and duodenum to the gall-bladder fossa.

DISCUSSION

The object of this set of experiments was to develop a method whereby the risk of operation upon the acutely inflamed or sclerotic gall-bladder would be diminished. The chief danger in these cases is hemorrhage, which treatment with cautery or electrosurgery helps to control. It is reasonable to suppose that if hemorrhage could be prevented with the markedly diseased gall-bladder it would be with the less damaged vesicle, but this does not appear to be true. The coagulating current, which is best for controlling the hemorrhage in extremely vascular tissue, must be selectively used with the gall-bladder for the best results. For instance, in treatment of thirteen non-sclerotic gall-bladders by the modified Pribram method, using the coagulating current, seven of the dogs died of hemorrhage from the operative site. Apparently the coagulated tissue of the vessel wall and that about it dissolved too rapidly. The uniterminal coagulating current is probably more offensive than the biterminal in this respect. It must be concluded that all the larger vessels should be ligated and the tissue in their immediate vicinity not coagulated.

Thorek[8, 9] has drawn a distinction between important types of electrocoagulation for cholecystectomy: Fulguration, or treatment by sparking without contact of the electrode, produces a more superficial, yet intense effect, resulting in carbonization. On the other hand, real electrocoagulation, obtained by inserting the needle into the tissue, or by holding a flat electrode in contact, leads to the formation of a firm coagulum, which, though more effective in disposing of the gall-bladder with little disturbance to the patient, will not dissolve so readily and lead to secondary hemorrhage. Even so, it seems preferable to tie the large vessels rather than to treat them electrosurgically by any method.

In these experiments results with electric current of different types were all better in the sclerotic gall-bladder—of seven dogs, none died from hemorrhage.

CLINICAL EMPLOYMENT OF ELECTROSURGERY FOR CHOLECYSTECTOMY

Since experimental results indicated that treatment of the severely inflamed or sclerotic gall-bladder with the coagulating current was a safe procedure, and since in these cases dissection by the ordinary method is rather hazardous, it seemed that the clinical use of electrosurgical cholecystectomy in selected cases was warranted.

This was accordingly carried out by the following technique, which was an outgrowth of our experimental observations: "After carefully packing away other viscera and placing self-retaining retractors the gall-bladder is aspirated and split longitudinally to the cystic duct, or near it, using the cutting current with high dehydration. Fluid and stones are removed by the aspirator and spoon. Then by the same means the tissue of the gall-bladder is cut away at about a centimeter from its attachment to the liver, the bleeding vessels being snapped and tied. The section of gall-bladder adherent to liver is not removed, avoiding danger of serious hemorrhage there. This tissue is thoroughly coagulated and dehydrated by fulguration. If any considerable portion of mucosa is left it may regenerate, resulting in a persistent sinus. If the cystic duct can be readily located it is tied or drained as indicated; if not it is ignored. The region is well drained, omentum being packed in to protect other viscera and prevent crippling adhesions. Stones may be left behind, either in the cystic or common duct, but this is better than risking death of the patient or doing irreparable damage in attempts at their immediate removal, since they can be handled by a secondary operation after the acuteness of the disease has subsided. The chief advantage of the method, however, is that since the main infective focus is removed, secondary operations are less often required than after simple drainage of the inflamed viscus."[10] Note the statement "this tissue is thoroughly coagulated and dehydrated by fulguration". The method employed was to treat the remaining tissue of the gall-bladder both by contact with the needle, and by sparking. This method was published before I learned of Thorek's work which took account of the difference between contact coagulation and fulguration. I am grateful to Thorek for the distinction, and wish to emphasize the

necessity for employing real contact coagulation in electrosurgical cholecystectomy. A second article dealing with results in clinical cases will be published later.

SUMMARY

1. The great advantage of electrosurgery is in control of hemorrhage. It has been shown by others that vascular tumors and organs, such as liver, kidney, and spleen, may be treated, first by deep coagulation, and then removal with the cutting current, without hemorrhage. Our experiments have confirmed this finding, large sections of the liver having been removed safely in this way.

2. As an outgrowth of Pribram's "*mukoklase*" a method was tried for destruction of the gall-bladder simply by cauterization of the cystic duct. This proved to be unsatisfactory on account of bile leakage and sloughing of the gall-bladder. Cauterization of the whole mucosa by the Pribram method in the non-sclerotic gall-bladder was likewise unsatisfactory. In the sclerotic gall-bladder it worked fairly well but we believe that any treatment of the gall-bladder by the actual cautery is too dangerous and difficult for general use.

3. The tissue-dissolving or "cutting current" was also too difficult of application for obliteration of the gall-bladder. Its use for "mukoklase" was unsatisfactory in the non-sclerotic gall-bladder, the chief danger being hemorrhage. With the sclerotic gall-bladder the results were better. However, it is not recommended.

4. The uniterminal coagulating current (desiccation) is too highly destructive of tissue, and the effect too superficial, to be safe. In our experiments its use was followed by secondary hemorrhage.

5. The method of preference finally developed: Aspirate the gall-bladder and split it to the cystic duct with the cutting current. The duct is dissected free and tied. The leaves of the vesicle are then trimmed away about a centimeter from the attachment to the liver, clamping and tying the bleeding vessels. Then the section of gall-bladder left attached to the liver is thoroughly treated with the coagulating current, by contact with the needle and fulguration, avoiding the points where the vessels are tied. The method of Thorek employing contact coagulation to considerable depth seems preferable to fulguration.

6. Drainage was not employed except in three of the experimental dogs. We have felt, however, that drainage is preferable in cases where there is considerable dead tissue to be absorbed. In the article* on clinical application of these experimental results (drainage being employed) the question will be discussed.

7. Electrosurgical cholecystectomy is best adapted to the markedly inflamed, or the sclerotic gall-bladder.

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ANTERIOR POLIOMYELITIS WITH REFERENCE TO THE OCCURRENCE OF TWO ATTACKS IN THE SAME INDIVIDUAL*

(With Report of Two Cases)

BY LOUIS COHEN, M.D.†

A SECOND attack of anterior poliomyelitis occurring in the same individual is very rare. To date there have been reported thirteen such proved cases. In over three thousand cases of anterior poliomyelitis seen in the Department of Orthopedic Surgery at the University Hospitals of the State University of Iowa there have been only two cases which have definitely represented a second attack of the disease.

Flexner, in his experiments on monkeys, could in no case produce a second attack of anterior poliomyelitis in an animal, once that animal had been infected and had survived. He injected the second dose of virus at intervals varying from eight days to four or five months after the onset of paralysis resulting from the first dose. In no instance was the monkey rendered ill or was there a recrudescence of the paralysis. He concluded that, "These results indicate what will probably be found to be equally true of human beings, that one undoubted attack of poliomyelitis, even when unaccompanied by def-

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inite paralysis, produces a state of refractoriness in reinoculation with active virus that endures for months and probably for years, if not throughout the life of the affected individual." He was also able to demonstrate by animal experiments that human beings who have been infected with anterior poliomyelitis and have survived contain in their blood for two or more years certain neutralizing principles for the virus causing the disease.

comparatively not rare. The second group comprises those cases in which the second attack occurs two or more years following the first attack, and until now only thirteen such proved cases have been reported. Only the latter group of cases can be considered as representing a true second attack and the former is explained as follows:

The immunity acquired by man as a result of one attack of anterior poliomyelitis occasionally

Case No.	Date report Author	Sex	Age of Attacks	Interval between attacks	Parts affected
1.	Caudouin 1879	M	1) 17 mos. 2) 16 yrs.	14 yrs.	1) lt. leg 2) rt. leg
2.	Ballet and Dutil 1884	F	1) 3 yrs. 2) 12 yrs. 3) 14 yrs.	9 yrs. 2 yrs.	1) lt. leg 2) arms 3) legs
3.	Eckert 1911	Not ment.		5 yrs.	1) lt. leg 2) rt. leg
4.	Lucas and Osgood 1913	M	1) 2 yrs. 2) 5 yrs.	3 yrs.	1) feet & rt. leg 2) rt. arm; legs weak
5.	Sanz 1915	F	1) 1 yr. 2) 15 yrs.	14 yrs.	1) lt. leg 2) rt. leg
6.	Taylor 1916	M	1) 3 yrs. 2) 6 yrs.	3 yrs.	1) rt. leg 2) lt. leg
7.	Francis and Moncrieff 1919	F	1) 3 yrs. 2) 18 yrs.	15 yrs.	1) rt. arm 2) legs
8.	Peremans 1923	F	1) 2¾ yrs. 2) 4¾ yrs.	2 yrs.	1) lt. leg 2) one arm
9.	Still 1930	F	1) 1¾ yrs. 2) 7½ yrs.	5¾ yrs.	1) lt. leg 2) rt. should.
10.	Neal 1932	F	1) 4 yrs. 2) 8 yrs.	4 yrs.	1) rt. leg 2) rt. leg
11.	Comm. Study of Inf. Par. Baltimore 1932	M	1) 16 mos. 2) 21 yrs.	20 yrs.	1) lowers 2) lt. thigh
12.	Quigley 1934	F	1) 5 yrs. 2) 7 yrs.	2 yrs.	1) lt. sh. 2) died before onset
	To this must be added Moore's case, since reported:				
13.	Moore 1934	M	1) 10 mos. 2) 7 yrs.	6 yrs.	1) lowers 2) arms and trunk

Lucas and Osgood, in 1913, reported a case illustrating a second attack of acute anterior poliomyelitis. Four months following the onset of this second attack the virus present in the nasal secretion of the patient was of such active virulence that when injected intracerebrally into a monkey it produced a typical and fatal attack of poliomyelitis in that animal. This is to be kept in mind when we consider that there are two groups of recurrence of anterior poliomyelitis which are definitely distinct from each other. One group comprises those cases in which the second attack occurs within four months after the first attack and which are is slow in developing. The active virus may remain in the nasopharyngeal secretion of the individual for a period up to four months following an attack, as shown by Lucas and Osgood. Therefore, any relapse occurring within this period can be explained as an auto-infection of the individual by the active virus present in his own nasopharyngeal secretions, this occurring before the full development of immunity following the first attack. This does not, of course, completely eliminate the possibility that the second attack may be a reinfection from a new external source.

When the interval between the two attacks

is two or more years the reinfection is undoubtedly a new one from an external source, as the active virus has experimentally never been found to be present in the nasopharyngeal secretion of a patient later than four months after the termination of the acute febrile stage of the disease in such virulence as to reproduce the disease in a monkey. There have been no cases of a second attack reported, whether it be a recrudescence or a reinfection, occurring in the interval between four months and two years covery following the first attack.

It is thus logical to conclude that immunity to anterior poliomyelitis once it is fully developed, either as a result of an attack or a recrudescence of an initial attack, is absolute only up to a period of two years.

The thirteen proved cases of reinfection with anterior poliomyelitis in the literature are shown in the large table, taken from Quigley[17] and Still[16].

In no case was there reported a complete recovery following the first attack.

To the table, reporting cases 1 to 13 inclusive, must also be added the author's two cases.

The following are the two cases seen in this clinic:

CASE 1:

H. L. D., a thirty-one year old married white male, came to the Out-Patient Clinic on January 5, 1934, complaining of general debility and weakness in the region of the left biceps, both of four months' duration. He stated that he had had infantile paralysis involving the right arm and right leg in 1908 and had, at that time, remained off his feet for one year, following which he used crutches for one year. The disease was epidemic at that time. Following this he improved. He retained some residual paralysis but has been able to get about without the aid of a cane or crutches. He worked in a garage and could do a full day's work easily until September 2, 1933, when he had what was diagnosed as a gall bladder infection and was put to bed for three days. When he got up his legs and arms were so weak that it was necessary for him to go to bed again and remain there for two weeks. He had difficulty in moving his arms and legs. He had pain in his neck and back, ached all over, had some elevation in temperature, did not eat well for one week, but did not vomit. His strength has gradually returned but he is still unable to walk without the aid of a cane.

He states very definitely that the left arm was not affected by the first attack and that the right leg is now somewhat weaker than before the second attack, and that the right arm was not affected by the second attack.

His family history and marital history were noncontributory. His past history was also negative, except as mentioned above.

His physical examination was negative except for the following:

He walked with the aid of a cane.

He had a moderate lordosis, ankle valgus on the left, equinus of the right foot, and two inches shortening in the right lower leg.

Reflexes: Patellar reflex, reinforced { Rt. absent. / Lt. slight.

Tendo-Achilles, reinforced { Rt. absent. / Lt. slight.

Case No.	Date report Author	Sex	Age of Attacks	Interval between attacks	Parts affected
14.	Cohen 1935	M	1) 6 yrs. 2) 31 yrs.	25 yrs.	1) rt. arm rt.leg 2) lt. arm rt. leg weak
15.	Cohen 1935	F	1) 12 yrs. 2) 30 yrs.	18 yrs.	1) rt. arm lowers 2) arms, legs trunk

Sensations were normal.

The muscles of the left leg were normal. There was practically complete paralysis of all the muscles of the right leg and there was marked atrophy of both the right thigh and calf. There was a moderate lordosis and a positive Trendelenburg test on the right.

The muscles of the upper arm showed considerable paralysis bilaterally; those of the lower arm were unaffected.

In this patient an arthrodesis of the right shoulder was indicated. He did not desire an operation and was discharged until such time as he might consent to one.

CASE 2:

E. K., a thirty-year old white married female, entered the hospital because of inability to use the right arm and both legs. She was seen in the orthopedic Out-Patient Clinic on January 7, 1935. She states that she was perfectly well until November 4, 1934, except for a very slight sore throat during the three to four days preceding that date. On the following afternoon (Monday) her right shoulder ached and her neck was stiff. She had no fever and continued with her usual activities. On the next day (Tuesday) her legs began to ache. On the following day (Wednesday) her "right arm became completely paralyzed". The legs continued to ache and were "weak" but she was able to be up and continue with many of her usual activities. She called an osteopath that night who told her that she had "neuritis" and gave her a salve. She remained in bed and the next night she called a physician who told her she had "infantile paralysis" (Thursday, November 8).

Her physician states that she then had a temperature of 104°, pulse 136. He further states, "There was practically a complete paralysis of the right arm and a general weakness of the left arm and both legs. The reflexes were absent or very weak."

Two days later her legs became completely par-

alyzed. She could, however, move her feet. Two to three days later the left arm became paralyzed. She remained in bed for three weeks. During this period the pain in the right shoulder was at times severe enough to require codeine for relief and the pain in the legs gradually disappeared. She has had no pain since then. The left arm improved considerably during the first week and has been improving gradually since then so that she could, on admission, practically feed herself. The right arm remained about the same until two weeks before admission since which time it has improved only slightly. The right leg has improved to about the same degree as the right arm. The left leg has shown no improvement.

During the three weeks prior to admission she also received some massage.

She was admitted to the neurological service on January 3, 1935. There lumbar punctures were done, a diagnosis of anterior poliomyelitis was made, and she was referred to the orthopedic service.

She knows of no other case of anterior poliomyelitis in her neighborhood.

At no time did she receive serum.

She states that she had infantile paralysis eighteen years ago (October, 1916), with involvement of both legs and her right arm. This is corroborated by the physician who treated her at that time. "She could not lift her right arm or right leg off the bed, and could barely raise the left leg." There were two other cases in the immediate neighborhood. She received no serum.

At present her main disability is inability to use the right arm and both legs.

Marital history, family history and past history are non-contributory except as mentioned above.

Physical examination revealed a young female whose only positive findings were as follows:

The knee jerks, ankle jerks, and biceps, triceps and radial periosteal reflexes were absent bilaterally.

The right arm and both legs were almost totally paralyzed. The left arm also showed marked involvement of a considerable number of muscles. There was also a marked involvement of the trunk.

This patient has been receiving bed rest and physical therapy, also splints to prevent the development of deformities.

The interval between the two attacks in the first case here reported was twenty-five years. The recovery following the first attack was incomplete. Although the right leg, in which there was a residual paralysis following the first attack became somewhat weaker following the second attack, the part affected most in the course of this second attack was the left upper arm, which had not been affected in the first attack. Thus we see that, except for the fact that the interval between the two attacks in this case was longer than the interval in any of the other cases reported, it presents no feature unusual to them.

The second case here reported does, in some respects, differ from any of the other cases reported. First, there was a more extensive involvement as a result of the first attack than in any of the other cases, i.e., total paralysis of two extremities and almost total paralysis of a third. Secondly, in no other case was there a complete recovery following the first attack. Thirdly, the three extremities which were affected as a result of the first attack were affected to practically the same degree during the course of the second attack. The fourth extremity, i.e., the left arm, which was also completely paralyzed during the course of the second attack and which was not affected during the first attack, is the only one which has recovered to any appreciable degree.

CONCLUSIONS

1. It has been impossible to produce a second attack of acute anterior poliomyelitis in monkeys. It is thought that one attack confers upon them a lasting immunity to the disease.

2. There are two groups of recurrence of anterior poliomyelitis in man which are definitely distinct from each other. One group is due to a recrudescence of the infection, occurs within four months of the first attack, is not rare, and is not a true reinfection. The second group is a true reinfection, occurs two or more years after the first attack, and is rare.

3. Immunity to anterior poliomyelitis, once it is fully developed, is absolute only up to a period of two years.

4. Two cases are reported here, one of which presents unusual features.

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TORSION OF THE PREGNANT UTERUS

Report of Two Cases

BY HILBERT F. DAY, M.D.*

TORSION of a pregnant uterus is undoubtedly rare and may cause a real surgical emergency. It is my object in the following pages to bring the literature up to date and to report two personal cases.

A study of the literature shows that the largest number of cases reported have been by French and German physicians rather than in English periodicals. I have found that the most satisfactory English article on the subject is that by Robinson and Duvall, published in 1931 in the *Journal of Obstetrics and Gynecology* of the British Empire. Their review is so complete and their abstracts of twenty-five cases by other writers so satisfactory that there is no reason for the writer to review the articles they have abstracted. Therefore, I am accepting their bibliography of fifty-two references and adding to it about thirty new references with abstracts of twelve.

General Statement: Torsion of the pregnant uterus has for many years been reported in journals having to do with veterinary obstetrics and is not a too rare complication of a cow's labor. In the human being it has been more recently reported and is far more rare. Many writers in speaking of torsion of the uterus have described the mechanics underlying this accident and have reported some cases which seemed to have occurred in a normal uterus, but from my experience and from examining the literature, I am inclined to feel that the uterus does not become twisted unless there is some abnormality in it. Symptoms may be absent, slight, or very acute, according to the rapidity with which the torsion occurs. Unquestionably, the most common causes of torsion of a pregnant uterus are the following:

1. A fibroid or fibroids.
2. An ovarian tumor.
3. The gradual contraction of a chronic inflammatory process on either side of the uterus.
4. Postoperative scar contraction.
5. A combination of the above causes.

Practically all of the cases the author has been able to review have shown torsion toward the right. It is difficult to state what starts torsion in these cases, for the history of most of the cases does not give this. Some authors think that the twisting from left to right is more common because of the fact that the descending colon fairly well fills the space on the left side. It is possible to conceive that some sudden movement in a case which has one of the predisposing causes might start the rotation. In one of the author's cases, it followed a too long automobile ride.

Diagnosis: In practically none of the cases described in the literature was the diagnosis of torsion of a pregnant uterus made before operation or death. Truly this rare disease has no single characteristic symptom on which to base a competent diagnosis. The surgeon who knows of this condition should at least consider it among others when he has to treat a pregnant woman, suffering from an acute abdominal crisis, whose condition is obscure. To make such a diagnosis, there should be a careful general history, menstrual history, description of recent symptoms, a complete general physical examination, including rectal examination, and ordinary laboratory findings, particularly a white count. Most important of all is a painstaking gentle abdominal examination.

History: The chief symptoms which recur often in the cases reported in the literature are the following:

1. Abdominal pain:
   a. Gradual onset increasing to such severity that only a large dose of morphia will relieve it.
   b. Sudden pain most generally localized on the right side of the abdomen, which may be so severe that only a large dose of morphia will give any relief.
2. Shock:
   In some cases the shock is so severe as to cause collapse. It is very difficult to differentiate this and shock of a sudden peritonitis or a partially separated placenta.
3. Abnormal findings:
   a. The uterus may seem of atypical shape. For instance, a large fibroid on the side may feel like an additional fetal head when partially obscured by a very tense abdomen.
   b. The abdomen has not the ordinary resistance of general peritonitis, but is extremely tense. Tenderness is not uniform over the abdomen, but is more marked above the pubes and on the side toward which the torsion has occurred. With the administration of a little ether, the signs of collapse recede, and the abdominal spasm dis-

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appears. Then the uterus and possible fibroid can more easily be defined.

4. No external hemorrhage:
   Practically nobody has reported any vaginal bleeding in these cases. Occasionally there has been found some internal hemorrhage.
5. Tenderness on rectal examination:
   a. Generally, a hard cervix is felt with tenderness in the vault.
   b. All necessary information can be obtained by a rectal examination.
   c. The author thinks that cases revealing the above recorded symptomatology should not have a vaginal examination because of the probability of a laparotomy and the possibility of either carrying infection up into the vaginal canal or stimulating labor by examination of the cervix.

Treatment: The author believes that manipulation in an attempt to reduce torsion is usually unsuccessful and therefore unwarranted and that operative interference should be resorted to as speedily as possible. As said before, the condition of shock seems to be relieved as soon as the anesthetic is started and he believes that the cause of a torsion can only be relieved surgically. What can be or should be done at the time of operation can only be determined by what the operator finds. If it is possible to do myomectomies and then reduce the torsion, as in one author's cases, that method should be practiced. It may be necessary besides this to do a hysterotomy or section; a hysterectomy if the uterus does not seem to be viable.

The above statement that manipulation is unwarranted, is not absolute as is shown by the following case:

Dr. David Rose, of Cambridge, told me that he was called to see a woman at the end of the first stage of labor, who was suddenly seized with a severe abdominal pain and was in beginning shock. The doctor who was attending her told him that during her pregnancy he had noticed a fibroid on the left side of her uterus. On examination, Dr. Rose found this fibroid on the right side and by manipulation was able to shift it back to its original position. The patient's pain disappeared and she delivered spontaneously a few minutes later. This undoubtedly was another case of torsion of the pregnant uterus in its initial stage.

REPORT OF AUTHOR'S CASES

In the following paragraphs, I wish to describe two personal cases of torsion of a pregnant uterus. The first case presented no problem but proved to be a curiosity. The second case presented a very difficult diagnostic problem and entailed a moderately serious operation. The twisting of the uterus generally takes place from left to right, as it did in both of my cases. The most common cause of such twisting is due to a tumor attached to the fundus of the uterus.

CASE 1. Mrs. M. C. was operated on January 6, 1922 at a small hospital in the suburbs of Boston. She was a twenty-two year old Jewess, 4 ft. 10 in. tall, weighing 105 lbs., who had never had any menstrual disorder. She had been married three years. She had a justo-minor pelvis and had been previously delivered by Caesarean section in a Boston hospital after fourteen hours of ineffectual labor. The baby of that delivery died at the hospital.

Despite her previous Caesarean section and the fact that her measurements were so low as to be absolute indications for delivery from above, plus a good-sized baby in this pregnancy, she was allowed to have twenty-four hours of labor and, previous to my seeing her, had had a vaginal examination.

OPERATION: Perpendicular abdominal incision to left of umbilicus four inches long, two-thirds below the level of the umbilicus. Abdomen opened, uterus inspected and the scar of the previous Caesarean section could not be found, but a tube and ovary were seen slightly to the right side of the midline of the uterus. The uterus was of normal color and it was impossible to rotate it one way or the other, and, therefore, although I suspected that the posterior surface of the uterus was presenting, I opened it through that surface and delivered a healthy seven-pound boy. The uterine wall was closed in three layers after the delivery of the placenta. Then on examination after some shutting down of the empty uterus, I found that there was a firm band of scar tissue from the anterior surface of the uterus to a point low down in the right pelvis. This I separated between clamps and tied off, and then discovered that the torsion of the uterus had been caused by a large ovarian cyst which had fallen into the pelvis. This tumor was removed. (The histological examination reported by Dr. William A. Hinton of the Harvard Medical School was as follows: "Histologic examination shows tissue of mesothelial origin presenting no evidence of malignancy.") After the removal of the band and tumor, the uterus returned to a normal position, rotating from right to left side. My line of uterine sutures entirely disappeared as they were through the posterior surface of the uterus. This patient's puerperium was normal. The patient was examined two months later by me and was found to have a strong scar and to be in excellent physical condition.

CASE 2. Mrs. C. was seen in consultation in a hospital during August, 1933 at the request of Dr. S. M. As the patient was in considerable shock, the following history was obtained from him and the patient's husband. She had been married three years and had had no pregnancies, and during that period had led a very active life. In the past month she and her husband had taken an automobile trip in Nova Scotia. She was perfectly well up to four days previously, when she had some abdominal pain and consulted a doctor in St. John, N. B., who advised her to get home quickly. Following his advice, she returned by the next boat instead of driving down. Immediately on getting home she called a doctor who found her in bed complaining of such severe abdominal pain that he gave her ½ gr. of morphia. This doctor reported to the physician with whom I saw her that at the time of his examination the abdomen was fairly soft, but that he felt a larger mass slightly above the level of the umbilicus on the right side. He described it as a tumor the

size of a full term baby's head. He said he thought of pregnancy particularly because the patient's husband said that he thought his wife's last period was about four months ago, but that he did not find any signs in the breasts to substantiate this idea. He made no vaginal examination. The next day Dr. S. M. saw her and, finding her in a serious condition, took her immediately to a hospital where I examined her with him.

When I examined the patient in the hospital, I found a woman in shock, who looked very sick and was actively nauseated. Quite evidently she was suffering considerable abdominal pain, particularly in the lower abdomen. She complained considerably of this pain and said that it was much worse if she had to lie on her back; therefore, she preferred to lie on her right side. Her temperature was 98.4°, her pulse 120 of rather poor quality, her leucocyte count was 20,000, systolic blood pressure was 102. A catheter specimen of urine was found to be normal.

The patient was a well-developed and nourished woman, weighing 145 lbs. and was 5 ft. 8 in. tall, blonde type. Eyes, ears, throat normal. Tongue was slightly coated. Heart area normal, action regular, rapid, with no murmurs. The lungs were clear. Breasts seemed to me to be slightly enlarged and to suggest pregnancy, although no fluid could be expressed from the nipples. The abdomen was distended throughout, rigid, tense and tender, and there was a suggestion of a mass to be felt in the right upper quadrant. A pelvic rectal examination revealed a slightly softened cervix with a tender mass in the posterior culdesac.

Because of the patient's general condition and the above findings, it was decided that she had an abdominal condition warranting immediate surgical investigation.

Preoperative diagnoses:

a. Pregnancy plus ovarian cyst with twisted pedicle.
b. Pregnancy plus fibroid with twisted pedicle.
c. Extra-uterine pregnancy ??

OPERATION: Patient was given morphia grs. 1/4 and atropine grs. 1/100 immediately and taken to the operating room after emergency preparation. In the operating room the patient was given ether anesthesia and, as is the case in so many patients in shock, as soon as she became anesthetized, the condition of her pulse improved and the abdominal spasm let up. We were then able to palpate the abdomen and we felt a large mass on the right side slightly above the level of the umbilicus which we had been unable to feel definitely with the abdomen rigid. Ether, iodine, alcohol preparation to skin. An incision about 8 inches long was made, running from pubes upward to 1½ inches above the umbilicus, slightly to right of midline. On opening the abdomen, there was an escape of considerable abnormal, straw-colored fluid. The examining hand showed that the mass previously felt was a fibroid tumor as large as a cocoanut, intimately connected with the uterus, not really pedunculated, although the base was about one-half the size of a cross section of the tumor. The uterus itself was found to be about the size of a four months' pregnancy, and the uterine muscle seemed slightly darker than normal in color. On careful examination it was found that the fundus was twisted halfway about to the right, and that, when this was corrected, the fibroid described above had arisen from the left side of the uterus. The mass previously felt by pelvic examination in the posterior culdesac was another fibroid about the size of a navel orange attached to the uterus which had been forced down into the pelvis when the uterus was twisted to the right. It was, undoubtedly, an additional cause for the patient's pain. After short deliberation, it seemed best to try to do myomectomies rather than a hysterectomy. The large fibroid was removed first. In so doing we cut down almost to the endometrium and there was considerable difficulty in sewing up the large defect. Three layers of sutures were employed and the bleeding finally controlled. The first two layers of sutures were continuous; the last ones were interrupted figure of eight sutures, No. 2 chromic catgut being used. The smaller fibroid from behind the uterus was removed in the same manner. After the removal of the tumors we kept the uterus under observation for about ten minutes to see whether there would be any serious bleeding. There was some oozing, but, as it seemed to be gradually diminishing, we decided to close the abdomen. The abdominal wall was closed in layers in the usual manner. Patient left the operating room in fair condition. (The pathological report of the tumors removed was "Leiomyomata.") For the first four or five days postoperatively the patient was kept absolutely quiet with morphia. After that, narcotics were omitted, and the patient made an uneventful hospital convalescence. The abdominal wound healed by first intention and the patient was out of bed on the thirteenth day. On this day she first felt life.

Before the patient left the hospital, she was advised to return for Caesarean section in January. That type of delivery was recommended because of the fear that large defects in the uterine wall repaired during a well-advanced pregnancy might leave a weak place in the uterus with the possibility of rupture during active labor.

On January 10, 1934, a classical Caesarean section was performed and a large, healthy, male baby, weighing 7 lbs. 14 oz. was delivered. When the abdomen was opened, some omental adhesions were found at the site of the myomectomy scar on the left side of the fundus, but the scar itself seemed fairly strong. The postoperative course following this Caesarean section was essentially normal, the only variation being a rather sharp postoperative hemorrhage about ten hours after delivery.

I believe the two cases reported above are of interest. Both are cases of torsion of the uterus from left to right. The first one apparently occurred so gradually as not to have caused pain, and the second so rapidly as to have caused very severe pain. I also believe that in the second case, if it had been impossible for me to control hemorrhage after the myomectomies, an emptying of the uterus by section, plus myomectomies, should have been considered rather than a hysterectomy, since the uterus was evidently viable.

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1. Bulletin De La Societé D'Obstetrique et de Gynecologie, De Paris. No. 9, November, 1932. P. 713.
Myome rouge à évolution rapide avec torsion axiale de l'uterus dans les suites de couches.
Trillat's case: A twenty-three year old woman with torsion of the uterus occurring three days after delivery. At operation no tumor found. Subtotal hysterectomy performed. Recovery.

2. Same Bulletin: No. 8, October, 1932. P. 574.
Torsion d'un volumineux fibrome au cours de la grossesse, M.M. Hamant et Hartemann.
Andre's case: A sixty-seven year old woman, always regular, examined 1931 before her marriage and found normal. Period February 14. In March had some pain. Operation in April. Fibroid tumor found turning uterus to the right. Myomectomy and interruption of pregnancy. Stormy convalescence. Final recovery.

3. Brussels Medical Journal: De Neve and Ingelbrecht. September, 1927.
Pedunculated Fibroid During Pregnancy: A twenty-seven year old woman, pains in stomach, two normal deliveries before. Last period May 15, that is, four months before. For four days pain in the flank. Mass in hypogastrium, pain on pressure. Day after admission operation. Serous fluid in the abdomen, peritoneal reaction. Fibroid size of new-born child's head from anterior surface of the uterus. Found behind the uterus. Myomectomy. Recovery. Subsequent normal delivery.

4. Bulletin De La Societé D'Obstetrique et de Gynecologie, Paris, 1929.
A forty year old woman between seven and eight months' pregnant. Axial torsion of the uterus due to a cyst on the right ovary. Symptoms: violent abdominal pains at the right of the abdomen with vomiting. Removal of cyst. Caesarean section. Female baby was feeble and died following operation. Woman also showed some fibroids in the uterus. Following operation she developed a pelvic phlebitis and died. Authors discuss relative merits of myomectomies and hysterectomies in such cases.

5. La Semana Medica. Buenos Aires.
A twenty-nine year old female, fifth month of pregnancy. Abdominal pain at navel. Torsion of the uterus found to be caused by a fibroid tumor. The twist untwisted as soon as the tumor was removed. Recovery.

6. Zentralblatt für Gynakologie. **54:** 2009, 1930. (Article by Litten.)
Excellent photograph of a woman with torsion of the uterus and radiographs of the same patient showing fetal bones and review of eleven cases.

7. Zentralblatt für Gynekologie. 1929. Wiegels.
A brief statement of a case operated by Dr. Wiegels where there was torsion of the uterus to the right about 90 degrees, held in place by adhesions. He feels that a diagnosis in such a case can only be made by operation or autopsy.

8. Monatsschrift für Geburtshülfe und Gynakologie. 1929. Berlin, Koerner.
Koerner reports a case of torsion of the uterus, rather gradual onset, torsion 90 degrees to the right. Operation and recovery of mother and child.

9. La Semana Medica, published in Paris by Drs. Enriquez.
Associated discussion of the general subject of axial torsion of the pregnant uterus produced by fibromata.

10. Revue de Gyn. et de Chir. Abdominale. Piquand and Lemeland. 1909.
An extensive discussion of the whole subject of torsion of the uterus particularly as due to fibroids of the uterus. The authors say that such torsion when in the course of pregnancy is rare, that they generally occur between the third and fourth months, that the onset is sudden, accompanied by severe pain and signs of peritonitis. Generally differential diagnosis. Immediate intervention is indicated. The treatment of torsions of the uterus in pregnancy should be handled in a way to preserve life and to permit pregnancy to go to term if possible. (Very comprehensive article on the whole subject.)

11. American Journal of Obstetrics and Gynecology. 1929. Article by Peightel.
Discussion of the fibromatous uterus, and he says that pregnancy does not seem to be an important etiological factor in causing torsion. Of eighty-four cases (Piquand's cases only) only fifteen occurred during pregnancy and eleven of these were during third and fourth months of pregnancy. He says that authors quoted in his article removed sub-serous fibroids and quotes five cases which continued pregnancy without interruption. He further says that if it occurs in the latter months of pregnancy, that Caesarean section with hysterectomy is the method of choice.

12. American Journal of Obstetrics and Gynecology. 1930. Article by Feiner and Kaldor.
Short discussion of the subject. They mention the fact that Stork and Hogt believe that operations performed some time previously have brought about unusual fixation of the uterus in certain cases. They report a case: Italian nullipara, thirty-eight years, entered clinic in labor with transverse position of the fetus, very little dilatation of the cervix. Because of lack of progress, intense pains, transverse position, laparotomy was decided upon. Uterus found rotated 160 degrees to the right. Caesarean section—vertical incision made on the posterior surface, after which the rotated uterus was returned to normal position, and the tremendously dilated varicose veins collapsed and became normal. No cause for the rotation discovered.

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POSTOPERATIVE GAS PAINS

BY EDWARD J. OTTENHEIMER, M.D.*

POSTOPERATIVE gas pains represent, unquestionably, the most common complication of major surgical procedures. Yet, as Ochsner[1] recently pointed out, their importance does not seem to be universally appreciated, nor have they been accorded the thoughtful consideration which their frequency and significance warrant. This may be explained by the fact that they are seldom serious in their consequences and are usually transitory in character, rarely lasting beyond the fifth postoperative day. Nevertheless, few surgeons will deny the sense of relief which accompanies the termination of the gas pain period, and all will admit that it is, disturbingly often, the one unpleasant memory which the patient associates with his surgical experience.

It is a matter of common knowledge that an extensive operative procedure may be followed by a smooth convalescence absolutely devoid of gas pains, while a simple, uncomplicated appendectomy may give rise to gas pains of such severity as to cause the surgeon apprehension and even alarm.

This variability in the appearance of gas pains has caused considerable speculation concerning the factors which might play some rôle in their production. In some instances, certain factors have been suggested as the result of laboratory experimentation as well as clinical observation, while in other instances the causative factors have been adduced simply as impressions based upon individual experiences.

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Among the most widely recognized factors and impressions are the preoperative régime, the length of preoperative hospitalization, the type of patient, the anesthesia, the duration of operation, the location of operation, the amount of trauma at operation, and the postoperative régime.

With the view of determining, if possible, the relative importance of these factors and impressions, a study of postoperative gas pains was undertaken. For this purpose it was considered fortunate that the Windham Community Hospital has no internes, since an exceptional opportunity was thus afforded to make personal bi-daily visits and observations. Special gas pain charts were prepared, a copy of which is submitted below:

NAME..... AGE..... HOSP. NO.....
OPERATION..... ANESTHETIC.....
DURATION OF OPERATION.....
TIME IN HOSP. PRECEDING OPER. .....
TYPE OF PATIENT.....
BOWELS PRECEDING OPERATION.....
PREOPERATIVE MEDICATION.....
AMOUNT OF MANIPULATION.....
POSTOPERATIVE TREATMENT.....
EFFECT OF MORPHINE.....
FLUIDS .....
GAS PAINS: DAY OF OPERATION.....
FIRST DAY POSTOPERATIVELY.....
SECOND DAY POSTOPERATIVELY.....
THIRD DAY POSTOPERATIVELY.....
FOURTH DAY POSTOPERATIVELY.....
OTHER DAYS POSTOPERATIVELY.....

As many of the data as could be were dictated preoperatively and in the operating room, and the rest given daily after the morning visit. A total of 400 consecutive major operations was studied.

TABLE 1

Gas Pains	Number	Per Cent
Patients having Gas Pains	236	59
Patients having No Gas Pains	164	41
Total	400	100

Table 1 shows that of the 400 cases studied, 164, or about forty-one per cent, had no gas pains, while 236, or about fifty-nine per cent, had gas pains.

In each case having gas pains, however, an opinion was ventured as to whether they were mild, moderate, or severe. Cases were classified as mild in which there were occasional cramp-like pains (not including the soreness which is associated with any incision), not accompanied by nausea, vomiting, or distention, and relieved by the unaided expulsion of gas. Many of these were so mild that unless one was looking for gas pains they might have passed unnoticed and the convalescence regarded as quite uneventful.

Cases were classified as moderate in which the patient complained of definite distress from distention, intermittent pain, perhaps nausea without vomiting, and requiring rectal tubes and enemata for relief.

Cases were classified as severe in which there were pronounced distention, nausea and vomiting (not the postanesthetic nausea and vomiting), severe colicky pain, elevation of pulse rate, and which necessitated the use of rectal tubes, or enemata, hot stupes, gastric lavage or any of the many other remedies commonly employed.

Based on this classification, table 2 is submitted:

TABLE 2

Gas Pains	Number	Per Cent
No Gas Pains	164	41
Mild Gas Pains	121	30
Moderate Gas Pains	51	13
Severe Gas Pains	64	16
Total	400	100

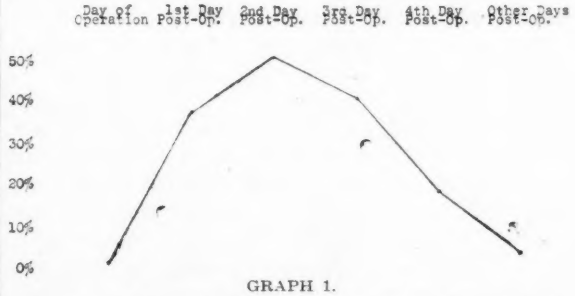
By grouping those patients having none, with those having only mild gas pains, as shown in table 3, figures are presented which probably more nearly represent the prevalent belief as to the frequency of postoperative gas pains.

TABLE 3

Gas Pains	Number	Per Cent
No Gas Pains or Mild Gas Pains	285	71
Moderate or Severe Gas Pains	115	29
Total	400	100

GAS PAIN PERIOD

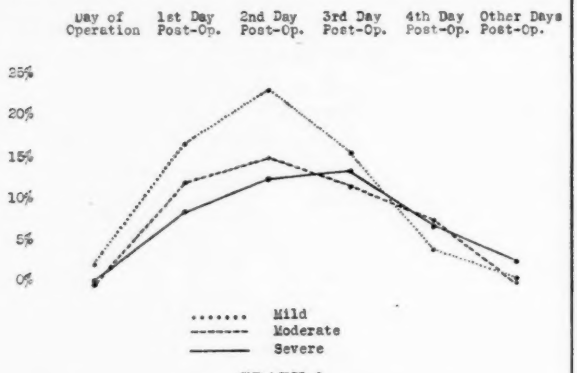
Based on the first 200 cases, graphs were prepared to show what may be called the gas pain period. Graph 1 shows that the period extends



GRAPH 1.

from the day of operation to about the fifth postoperative day. The incidence, however, on the day of operation and the fifth postoperative day, is extremely low, the highest incidence appearing on the second, third, and fourth postoperative days, with the peak on the second postoperative day.

In graph 2, the incidence is plotted separately for those having mild, moderate, and severe gas pains. This shows essentially the same as the above except that in the cases having severe gas pains, the peak is not reached until the third postoperative day, and the incidence on the fifth postoperative day is somewhat higher.



GRAPH 2.

While nothing was found in the literature to suggest that the sex of the patient had any influence upon the incidence of postoperative gas pains, it was felt that data on this point might be of some interest. Table 4 is therefore presented, from which the inference may be reasonably drawn that gas pains are just as common in males as in females.

TABLE 4

Sex	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Male	152	42.1	57.9	30.2	12.5	15.1
Female	248	40.3	59.7	30.2	12.9	16.5

THE FACTOR OF AGE

In a recent article, Victor Bonney[2] expressed the opinion that "postoperative functional derangement of the intestine" was more common in elderly than in young patients. When this study was undertaken, however, no written opinion as to the factor of age seemed available, and the data assembled in table 5 represented an attempt to determine if the age of the patient had any bearing whatsoever upon the incidence of "gas pains".

The number of patients operated upon below ten years of age and above seventy is obviously too small to be of any statistical value. It seems apparent, however, that the highest percentage of gas pains, almost seventy-two per cent, occurred in the seventy-one patients in the age group of forty to fifty. The highest percentage of severe gas pains, over twenty-three per cent, occurred in the age group between thirty to forty. It is obvious that in the age groups between twenty to fifty occurs the greatest incidence of gas pains. Table 6 is submitted to present a comparison of different age groups with the series as a whole.

TABLE 5

Age	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
1-5	1	100.	—	—	—	—
5-10	8	87.5	12.5	—	—	12.5
10-20	59	45.7	54.2	35.5	13.5	5.0
20-30	97	36.0	63.9	30.9	15.4	17.5
30-40	72	37.5	62.5	30.5	8.3	23.6
40-50	71	28.1	71.8	32.3	18.3	21.1
50-60	51	45.0	54.9	31.3	13.7	9.8
60-70	33	54.5	45.4	27.2	3.0	15.1
70-80	8	75.0	25.0	—	12.5	12.5
Total	400	41.0	59.0	30.2	12.7	16.0

TABLE 6

Age Group	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
1-20	68	51.4	48.5	30.8	11.7	5.8
20-50	240	34.1	65.8	31.2	14.1	20.4
50-80	92	51.0	48.9	27.1	9.7	11.9
Entire Group	400	41.0	59.0	16.0	12.7	30.2

THE FACTOR OF ANESTHESIA

The part played by the anesthetic in the etiology of postoperative gas pains has been discussed by many writers. O'Keefe[3] states, "I cannot agree with the majority of writers as to the importance of ether in the cause of gas pains. It is undoubtedly a contributing cause, but it certainly has a transitory effect." Potter and Mueller[4] mention the anesthetic as a factor contributing to postoperative distention. Cunningham[5] believes the anesthetic must be considered a factor in the causation of postoperative gas pains.

In table 7, the various anesthetics used in this series and the associated incidence of gas pains are presented.

It would appear from this table that the highest percentage of gas pains (72 per cent), occurred in the cases having avertin and ether. It may be observed also that in cases in which avertin and ether were used, there was the highest percentage of severe gas pains (25.4 per cent). However, it should be noted that the total number of these cases was only fifty-five.

It should also be pointed out that the next highest percentage of gas pains occurred in cases

in which spinal anesthesia was used, although here again only fifty-eight cases were in this group. This, however, is rather an interesting observation since there seems to be current an impression that gas pains are less common after spinal than after an inhalation anesthetic.

TABLE 7

Anesthesia	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Gas Oxygen	2	100.0	—	—	—	—
Spinal	58	32.7	67.2	39.6	13.7	13.7
Local	4	100.0	—	—	—	—
Sacral	2	100.0	—	—	—	—
Gas Oxygen Ether	267	42.6	57.3	29.2	12.3	15.7
Avertin and Ether	55	27.2	72.7	30.9	16.3	25.4
Paraldehyde and Ether	12	66.6	33.3	25.0	8.3	—

In order to show the incidence of gas pains in cases having inhalation, as opposed to non-inhalation, table 8 is submitted.

TABLE 8

Anesthesia	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Inhalation	336	41.3	58.6	29.1	12.7	16.6
Spinal, Local, Sacral	64	39.0	60.9	35.9	12.5	12.5

While the number of cases having non-inhalation anesthesia is comparatively small, the results suggest, at least, that postoperative gas pains may be just as common after the use of local or regional anesthesia as after general, and indeed in this series slightly more common.

THE FACTOR OF THE DURATION OF OPERATION

Despite the fact that there has been a definite trend away from the rapid and spectacular, toward slow and gentle surgery, the majority of writers still feel that long operations contribute generously to stormy convalescences and show a higher incidence of postoperative gas pains and distention. Obviously there is an implication here that the more protracted the operation, the greater the degree of intraäbdominal trauma, the longer the exposure of the peritoneal cavity, and the larger the volume of anesthesia administered.

In table 9 is shown the incidence of gas pains in the various time groups.

It is interesting to note that there was a lower percentage of gas pains in the operations lasting one and one-half to two hours or more, than in those lasting one to one and one-half hours.

TABLE 9

Time	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Under ½ hr.	13	46.1	53.8	30.7	15.3	7.6
½ to 1 hr.	148	45.2	54.7	31.0	9.4	14.1
1 to 1½ hr.	148	36.4	63.5	30.4	17.5	15.5
1½ to 2 hrs. or over	91	40.6	59.3	28.5	9.8	20.8

However, it might be significant to point out the progressive rise in the percentage of severe gas pains as the length of operating time increased.

THE FACTOR OF THE LOCATION OF OPERATION

In an excellent discussion of postoperative gas pains Cutting[6] states, "distention probably occurs more frequently and typically after operations upon the biliary tract, less frequently after operations in the pelvis, and still less frequently following operations on the gastrointestinal tract. Distention is rare following operations for inguinal hernia." Bonney states "The incisional site has an influence. Incisions situated above the umbilicus, or well out to the side of the abdomen, are less apt to be followed by intestinal derangement than those situated below it or in the middle line."

Table 10 is presented to show the incidence of gas pains in operations of different locations.

TABLE 10

Location	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Upper Abdom.	61	31.1	68.8	36.0	14.7	18.0
Lower Abd. inc. Append.	247	34.4	65.5	34.0	13.7	17.8
Extra-Abd.	52	76.9	23.0	9.6	3.8	9.6
Herniae	40	50.0	50.0	25.0	15.0	10.0

It is apparent in this series, that while the incidence of gas pains was higher in upper abdominal than in lower abdominal operations, the difference was comparatively slight. The low incidence of gas pains in extraäbdominal

operations, many of which were urological, is not unexpected.

It is interesting to observe that the occurrence of gas pains in hernia cases was not so rare as Cutting suggests, although it must be said that the total number is comparatively small and includes strangulated as well as non-strangulated, and femoral, ventral, and umbilical, as well as inguinal.

THE FACTOR OF PREOPERATIVE PREPARATION

The preoperative management of surgical cases has been discussed widely in the literature of the past decade, and the discussion has centered chiefly upon the advantages and disadvantages of preoperative catharsis. At the present time, in this country, the use of strong purgatives the night preceding operation has been practically abandoned.

No attempt was therefore made in this series to determine whether gas pains were more common after the use of preoperative cathartics.

TABLE 11

Bowel Movement	No. Cases	% Having No Gas Pains	% Having Gas Pains
Day of Operation	140	47.9	52.1
Day before Operation	249	37.3	62.7
Two or more Days before Operation	11	36.3	63.7

The cases were all placed on essentially the same preoperative régime, and in no case was a purgative given the night before operation. Emergency cases, as a rule, were given no enemata but a record was kept as to when the bowels had moved last. If the patient entered the hospital the day before operation, an enema was given the afternoon before operation in most cases. In some cases, an enema was given on the morning of operation. Cases admitted two or more days prior to operation were given a mild cathartic two days before operation, and then the usual enema on the afternoon before the operation.

It might be instructive to point out, as shown in table 11, that postoperative gas pains were less common in those cases whose bowels had moved, either naturally or by enemata, the day of operation.

TABLE 12

Time in Hosp. Preceding Op.	No. of Cases	% Having No Gas Pains	% Having Gas Pains
Emergencies	106	51.0	49.0
One Day	148	40.5	59.5
Two or more Days	146	34.2	65.8

There is some evidence in the literature (Cunningham)[5], to suggest that the longer the preoperative stay in the hospital, the less the incidence of gas pains. Table 12 is therefore submitted with data on that point.

It is evident that in this series the impression of Cunningham is not substantiated, since gas pains increased in direct proportion to the length of preoperative hospitalization. Curiously enough, the lowest incidence of gas pains occurred in emergencies, which might be reasonably supposed to have had the least amount of preoperative preparation.

THE FACTOR OF TRAUMA

Trauma, during the operation, is almost universally recognized as an important causative factor in the production of gas pains, and great emphasis has been repeatedly placed upon the gentlest handling of tissue, the avoidance of excessive manipulation of the intestine, the care with which gauze packs should be used, and the elimination of any act which might interfere, temporarily, with the circulation of any part of the intestinal tract.

It is admittedly difficult to estimate the amount of trauma done during any operation and table 13 is, therefore, submitted with some misgivings. However, it must be said that in each case, a conscientious opinion was given as to the extent of trauma inflicted, ranging from one to four plus. Operations in which there was excellent relaxation of the abdomen, in which the exposure of the operative field could be made easily and gently, which required few if any packs, and in which the intestines had been manipulated either not at all or to only a very slight degree, were graded as 1+. Operations in which the abdomen could not be consistently well relaxed, in which the bowels seemed to "boil" into the operative field, and in which excessive manipulation of the intestine, repeated packing and repacking, and unusual retracting force seemed necessary to secure adequate exposure of the operative field, and to complete the technical details of the operation, were graded 4+.

TABLE 13

Trauma	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
1+	172	50.6	49.4	29.6	6.9	12.7
2+	175	31.4	68.5	32.0	18.2	18.2
3+	48	37.5	62.5	29.1	14.5	18.7
4+	5	80.0	20.0	—	—	20.0

It is felt that these two extremes may be readily visualized, and that between these extremes, the amount of trauma represented by

2+ and 3+ may possibly be more clearly interpreted.

The number of cases which had a grade of 4+ is too small to be of statistical value, and the number having 3+ is relatively small. The sharp rise, however, in the incidence of gas pains in operations having a grade of 2+ or 3+ as compared with 1+ seems significant.

THE FACTOR OF THE TYPE OF PATIENT

The patient who is faced with the necessity of undergoing a major surgical operation, in general may be said to react to his predicament according to the stability of his nervous system. The calm, poised, phlegmatic individual accepts the decision with equanimity. The highly emotional, neurotic, unstable patient is filled with apprehension. Between these extremes may be exhibited varying degrees of nervousness which should be easily discernible to the surgeon.

In this series of cases an opinion was hazarded in each case, preoperatively, as to whether the patient was of the nervous or phlegmatic type. While it is obvious that this observation is personal and conjectural, it is felt that the margin of error will be readily understood.

TABLE 14

Type of Patient	No. of Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Nervous	203	29.1	70.9	31.0	16.2	23.6
Not Nervous	197	53.3	46.7	29.4	9.1	8.1

Table 14 shows that of the 203 patients adjudged as being of the nervous type, 70.9 per cent had gas pains as compared with 46.7 per cent of those who were thought not to be of the nervous type. It shows also that the incidence of severe gas pains was much higher in the nervous group. Granting the fallibility of deduction based on presumption, the discrepancy between the two groups is so striking as to justify the opinion at least, that the nervous temperament of the patient has much to do with his postoperative gas pains.

THE FACTOR OF THE POSTOPERATIVE RÉGIME

No attempt was made in this series to study the effects of various forms of treatment of gas pains. All patients, except as specified in table 15, were placed on a more or less routine régime which consisted of sips of water gradually increased after twelve hours, morphine practically every three hours for the first forty-eight hours, heat to the abdomen, and elevation of the head and foot of the bed. In about one-third of the cases, tap water was given rectally immediately after operation, and at four hourly intervals for forty-eight hours. Further orders were based, of course, on the requirements of the particular operation, and whether gas pains occurred.

TABLE 15

Régime	No. Cases	% Having No Gas Pains	% Having Gas Pains	%—Mild Gas Pains	%—Moderate Gas Pains	%—Severe Gas Pains
Routine	356	37.9	62.1	30.6	13.8	17.7
Ochsner	44	65.9	34.1	27.3	4.5	2.3

As may be seen in table 15, there were forty-four patients who were placed on the Ochsner régime. These were chiefly cases of peritonitis from a ruptured appendix, or ruptured ulcer, or cases in which resection of some portion of the gastrointestinal tract had been done.

For practical purposes the only difference between the routine and the Ochsner régimes was that in the latter no fluids were given by mouth for from forty-eight hours to several days. Fluids in these cases were given either by fractional intravenous injections of glucose, or by the constant intravenous method.

While the number of cases on the Ochsner régime was comparatively small, it seems noteworthy that only one patient of the forty-four had severe gas pains, and the incidence of gas pains in this group as compared with the routine group, was correspondingly low.

This bears out what Alton Ochsner has said repeatedly, that following laparotomy there exists an inhibition of intestinal movement and a loss of intestinal tone. If the intestinal tract is allowed to rest, it soon regains its tone, but if stimulated too actively by the administration of fluids by mouth, and to some extent by rectum, gas pains result.

COMMENT

A study of gas pains based on 400 consecutive cases is submitted. The data are often, of necessity, based on crude figures, which may lead to conclusions capable of a moderate refinement. The danger of drawing inferences from comparisons of results obtained in cases which themselves are not always strictly comparable, is acknowledged. Nevertheless, the material presented constitutes a conscientious effort to analyze what many surgeons are apt to mean when they say, "It is my experience that thus and so is the case." Bearing all this in mind, it seems not unreasonable to present the following conclusions:

1. Gas pains are just as common in men as in women.
2. They occur most frequently between the ages of twenty and fifty.

3. They may occur as often after spinal as after inhalation anesthesia.
4. Long operations tend to increase the incidence of severe gas pains.
5. Gas pains are only slightly more common after upper abdominal than after lower abdominal operations.
6. Gas pains are not infrequent following hernia operations.
7. Long preoperative hospitalization does not reduce the incidence of gas pains.
8. Manipulative trauma plays an important rôle in the production of gas pains.
9. The nervous type of patient is much more likely to have gas pains than the phlegmatic type.
10. Gas pains can be greatly reduced by giving no fluids by mouth for twenty-four to forty-eight hours following any laparotomy.
11. It is felt, by way of conclusion, that routine postoperative treatment should be abandoned. If the surgeon is aware of the relative importance of the various factors contributing to gas pains, it may be possible for him so to individualize his postoperative treatment as to have a minimum of gas pains.

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INCARCERATED BLADDER IN A SCROTAL HERNIA

Case Report

BY FRANCIS J. PETRONE, M.D.,* AND EDWIN VIEIRA, M.D.*

BLADDER herniations are relatively frequent, incarcerations less frequent, but incarcerations in the scrotum are found to be very rare. Because of the scarcity of such cases reviewed in the literature, a report of a case recently observed is hereby submitted for record with a survey of vesical hernias.

Vesical hernias are not uncommon as has been revealed by Erdman when he collected 8600 cases of hernias of various types and found that in four cases out of every thousand the bladder was involved in the hernia. Scrotal vesical hernias are however, rare, as Makkas in 1933 stated in a report of a case of a large bladder hernia on the right side. Quivy and Yvon also reported such a case in 1934. These workers were fortunate in making a diagnosis by cystogram, showing the greater portion of the bladder in the right scrotal sac. The anamnesis in their case revealed that the patient had a hernia since the age of fifteen, and that in later years he noticed that he began to urinate in two stages, first normally, and finally by manipulating the scrotum. Pfitzner in 1930 reported a right irreducible hernia in the scrotum. The bladder was freed and returned to the abdomen and the hernia repaired in the usual manner.

Bladder herniations as already stated are not uncommon. In 1923, 406 cases were collected from the literature and it was found that they occur twice as frequently in men as in women. In 359 cases 130 were on the right side, 103 were on the left side, five were bilateral, and forty-two were not specified as to side. Femoral vesical hernias were included in the series, forty-three were on the right and twenty-one on the left side, two bilateral and thirteen were not classified as to side. Other varieties of vesical hernias occurred, such as perineal, sciatic, ventral, and obturator; these types were however, very rare.

Preoperative diagnosis of bladder hernia is very low. In 347 cases the diagnosis before operation was made in only twenty-five cases, 279 were diagnosed during operation and forty-three recognized after operation. It has been noted that injuries to the bladder during the operation seem to be a very frequent incident. In 406 cases of vesical hernias 194 were injured during operation, nearly 50 per cent. This complication naturally aggravates the condition, especially if not recognized during the operation. Resultant fistulas lasted from one day to one year or more. The mortality has been found to be higher in those cases in which injury was not recognized during the operation.

REPORT OF CASE

Chief Complaint: Pain in the right lower quadrant for the last six hours radiating to the groin.

Family History: One son and one brother have large inguinal hernias.

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Past History: Patient has enjoyed good health for the past twenty years. Has no history of serious ailments, accidents, or operations.

Present Illness: The patient was perfectly well up to the onset of his present condition at 8 A.M. He got up and, on doing chores about the house, began to notice severe lancinating pains in the right lower quadrant which radiated down to the right groin. Severity of the pain became worse until he was obliged to call his physician for relief. The patient noticed that he had been constipated the last few days, consequently took an enema without effect save for the passage of some gas. He had noted some difficulty in passing urine but never any retention requiring catheterization, nor has he had dysuria or passed blood. With the onset of the present condition a lump appeared in the right groin and gradually became larger. This swelling was in the right scrotal sac. It was tender to palpation and non-reducible. This lump had appeared in the past without symptoms and was always readily reduced on pressure.

PHYSICAL EXAMINATION:
The patient was a large framed, obese Portuguese farmer weighing about 200 lbs., in evident distress with pain, ruddy faced but slightly cyanotic and dyspneic. Temp. 99°, pulse 55, resp. 20. W. B. C. was 15,000 and the differential showed 80 per cent polys. The Wassermann test was negative, and urine analysis showed a slight trace of albumin, a few leucocytes, a rare hyaline cast and rare red blood cells. The tongue was heavily coated, mouth hygiene was poor, and the throat was injected. The chest was barrel shaped, excursions small but equal. The lungs showed the presence of sibilant râles throughout, with increased resonance and diminished breath sounds. Heart sounds were snapping in quality, a soft systolic murmur at the apex was heard but not transmitted. A-2 was accentuated. B. P. 210/100. Abdomen obese. A large swelling the size of a small grapefruit was noted in the right inguinal area, extending down to the right scrotum. This mass had the feeling and resiliency of a tennis ball, and was very tender to palpation. The right lower rectus was rigid. The extremities showed no edema or deformities. Reflexes were normal. Patient was admitted to the R. I. Homeopathic Hospital September 15, 1934, and was operated on the same day.

Preoperative Diagnosis: Incarcerated Inguinal Hernia. (Right.)

OPERATION: The patient was not considered a good surgical risk, but surgical intervention was deemed imperative and avertin anesthesia supplemented with ether was used. In ten degree Trendelenburg position the operative field was prepared with benzene-tr.-iodine. A four inch long incision was made one inch above Poupart's ligament down to the pubic spine over the mass. This mass was located inside the scrotum and outside the external abdominal inguinal ring. The primary incision extended down to the external oblique fascia. All bleeding points were clamped and tied. A marked constriction band was cut just outside the external inguinal ring, and when this was done the sac which was the size of a large orange collapsed. The cord was isolated from the sac to which it was firmly bound by adhesions. The sac was peculiar, baggy and felt as though its contents consisted of omentum firmly adherent to the sac. This sac came directly out of the abdomen and did not follow the cord up the inguinal canal. It emerged from Hesselbach's triangle down through the external ring. By gentle dissection an attempt was made to get to the inside of the sac. It was disappointing to find that the bladder had been exposed and opened, evacuating about four ounces of clear straw-colored fluid. It was realized that this was an unusual case of incarceration involving the bladder in the right scrotum. The bladder was immediately sutured with catgut. The redundant sac or tissue was clamped, ligated and cut. The bladder was then pushed into place behind the pubis and several interrupted chromic catgut sutures placed from the conjoined tendon to Poupart's ligament. The external oblique fascia was sutured with continuous chromic catgut and the skin sutured with interrupted silkworm catgut. A dry sterile dressing was applied and a retention catheter was placed in the bladder for continuous drainage. The patient took anesthesia very well except for moderate cyanosis at various intervals, and the general condition at the end of the operation was considered fair.

Postoperative Diagnosis: Incarcerated Scrotal Hernia of the Bladder (Direct-Right).

Progress in the Hospital: On the fifth postoperative day the retention catheter was removed and he began to void voluntarily but with some pain which gradually cleared up during the next few days. His urinary output increased, his appetite was good, his abdomen was soft and elimination was satisfactory. For several days he was troubled with general urticarial eruption. This was felt to be due to a throat infection as the urticaria cleared up when the throat improved. He was discharged from the hospital October 2, 1934, improved. The patient urinated in normal amounts without pain, and stated he was feeling well.

Pathological Diagnosis: Hernial sac with fat tissue.

X-Ray Report of Bladder: Cystogram showed a large filling defect of the right side of the bladder, which diminished the capacity of the bladder to less than one-half. The defect was probably due to the repair of the hernia of the bladder into the scrotum. No part of the organ protruded below Poupart's ligament.

Progress of the Case Since Hospital Discharge: Examination of patient six months after discharge shows a well-healed right inguinal incisional wound with no recurrence of hernia. Patient has had no urinary symptoms of frequency or dysuria. He is feeling well and doing work about his farm.

COMMENT AND SUMMARY

From a survey of the literature incarceration of a bladder hernia into the scrotum is rare, while at the same time herniation of the bladder in various positions is not an uncommon finding in lower abdominal hernias. In conclusion, it is obvious that in repairing any hernia in the lower abdominal region the bladder should be constantly kept in mind. Injuries to the bladder in these cases are too frequent a complication and are not even recognized at the time of operation. More attention should be paid to the history and particularly to the genitourinary aspect of the history. Whenever suspicious or

doubtful as to the presence of this complication the bladder should be catheterized and a cystogram made.

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THE ARTERIOGRAPHIC COMPARISON OF THROMBO-ANGIITIS OBLITERANS AND ARTERIOSCLEROSIS*

BY EDWARD ALLEN EDWARDS, M.D.†

THE arteriogram of normal arteries shows three characteristics which are changed in both arteriosclerosis and in thrombo-angiitis obliterans. These characteristics of normal arteries have been noted by Allen and Camp as follows: first, the smooth and uninterrupted contour of their lumens; secondly, the direct course of the vessels; thirdly, the presence of no more than a minimum of collateral circulation[1].

Examination of arteriograms of diseased arteries show striking deviations from the normal. In both Buerger's disease and in arteriosclerosis one finds varying degrees of blockage of the lumen, tortuosity of the entire vessel, and an increase in the number and size of the collaterals. I have observed, however, in common with Leriche, Fontaine, and Frieh[2], and with Sgalitzer, that one can nevertheless distinguish between the pictures of the two conditions.

The arteriosclerotic vessel (figure 1) may show not only abrupt blockage of the lumen, but also multiple eccentric rounded filling defects in the segments of the artery above the blocked portion. This gives a serpiginous outline to the image of the vessel lumen. Secondly, the lesser arteries and sometimes the main artery may be irregular or tortuous in their course. Thirdly, the number of collaterals are markedly increased.

The arteriogram in Buerger's disease is a different one (figure 2). The totally blocked areas of lumen are usually more extensive. Above these totally blocked areas one usually finds a lumen of smooth outline, with absence of the eccentric filling defects noted in arteriosclerosis. Secondly, the arteries are less apt to be tortuous. Thirdly, the visualized collaterals are fewer in number than in the arteriosclerotic.

The difference between the irregular lumen of arteriosclerosis and the smooth appearing lumen of Buerger's disease, can be explained by the occurrence of localized atheromata in the former disease, and their absence, in uncomplicated cases, in the latter. While discrete areas of proliferative thickening of the vessel wall may occur in Buerger's disease, the process is more apt to be one involving the entire circumference of the lumen, giving the lumen a reduced but regular outline.

Furthermore, we know from clinical and postmortem observations that the blood stream tolerates localized atheromata well, thrombosis occurring late in arteriosclerosis. In thrombo-angiitis, however, even a small extent of involvement is quickly followed by thrombosis. In this way the arteriogram of an arteriosclerotic vessel may show atheromatous changes in outline, with or without occlusion; whereas in Buerger's disease it is almost an "all or none" proposition, i.e., as soon as the artery is involved more than a very little, it is apt to be thrombosed, and show abrupt loss of its lumen up to the normal part of the artery.

Concerning the fewer collaterals in thrombo-angiitis, it is possible that it is to some extent a matter of time. That is, if the duration of the process were as long as in the arteriosclerotic, the collaterals might gradually develop to the same extent.

Previous work by Lewis and Reichert[3], and by Horton[4], indicated that the collateral circulation is much richer in thrombo-angiitis obliterans than in arteriosclerosis. This finding is contrary to that of Leriche, Fontaine and Frieh, and to my own findings. I believe the conclusions of the former observers are open to question because their arteriograms were made from amputated limbs, while those of Leriche, Fontaine, and Frieh, and of mine, have been made during life.

CONCLUSIONS

The arteriograms of arteries injected during life show characteristics which are seemingly distinct for arteriosclerosis and for thrombo-angiitis obliterans. In arteriosclerosis the arteriogram may show complete occlusion of segments but in addition shows eccentric filling defects in the other segments. They may also show a more tortuous course than normally; and the collateral circulation is greatly increased.

In thrombo-angiitis obliterans the arteriogram shows a varying extent of thrombosis, with smooth-walled segments above the occluded segments. The course of the vessels is less likely

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FIGURE 1. Arteriogram of a femoral artery involved by arteriosclerosis (made during life and later confirmed by pathological examination).

For the technic of arteriography see the author's previously published articles:

1. Edwards, Edward A.: The status of vasography. New Eng. J. Med. **209:** 1337, 1933.
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FIGURE 2. Arteriogram of a femoral artery involved by thrombo-angiitis obliterans (made during life and later confirmed by pathological examination).

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to be tortuous; and the collaterals, though increased in number above the normal, are yet fewer than in the arteriosclerotic.

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ALLERGY TO LIFE: AN INTERPRETATION OF NEUROTIC CONSTITUTION*

BY ELI MOSCHCOWITZ, M.D.†

DISPASSIONATE observers are fully aware that the neuroses are not only distressingly abundant, but that they are increasing in the United States. This, in itself, although serious, is made more alarming because certain common, sometimes fatal, maladies which I believe are in whole or in part of psychogenic origin, are increasing with prodigious speed. I refer especially to hypertensive disease, Graves' syndrome and gastric ulcer. We cannot flatter ourselves that all this increase is only apparent and that it is due to finer precision in diagnosis.

It is agreed that a neurosis is the result of a conflict between the environment and the individual and that it represents in a large measure failure to achieve the security for life or the satisfaction of the lust for power or of love, that is instinctive in human kind. Normality of conduct and behavior may be defined as an equation in which the forces of the environment are in delicate balance with the forces within the individual. The determination of the cause of the increase of neurotic illnesses therefore depends upon the answers to the questions as to whether the environment has become more onerous or the individual more sensitive or both.

It would be idle to deny that our environment has grown more onerous. It did not require an economic depression to make this apparent. Long before this event, life was become increasingly complex and was attended by a swiftness of pace so great that the human mind and frame were finding it difficult to keep in adjustment.

It is beyond my purpose and capacity to analyze completely the forces that made up the complex of our modern swift-paced civilization. Briefly as I see it, these forces are in some measure economic but for the greater part are psychologic. The unequal and unjust distribution of wealth is but one of the economic forces involved. Sufficient recognition has not been given to the fact that the progress of civilization has brought in its train a host of evils. If one looks into the mouth of the gift horse of progress one finds that this progress has been material rather than spiritual; that it consists for the most part in inventions that enable us to do two things where we could do only one before, so that the life pattern has become more compact; a person today lives two lives within the space of one.

Of greater import, I believe, in the increased production of conflict is the enhanced sensitivity of the individual to life and it is to the analysis of this influence that I now turn.

The most important as well as the earliest influence which sensitizes the individual is the family, and of this hierarchy the mother is the most dominating member. One of the important contributions of the Freudian psychology has been the study of the effect of maternal influences on human behavior. The emphasis that has been placed on the so-called mother complex, whether it is hate or love, and its effect upon the sex life of the individual is hardly exaggerated. The range of influence is far wider than upon sexuality. Other instincts and senses may be affected and through these conduct and behavior.

The mother exerts the devastating influence which, to a large extent, sensitizes the individual by the mechanism of overprotection. Overprotection does less harm when the mother's fears are against physical insult or illness, but it is bad enough here because it may create the habit of introspection. The tragedy is when there is an overmastering fear of self-determination, which from birth begins a desperate mental moulding, a mental transference that is unrelaxing. It is because of the fear of self-determination that some mothers cultivate within the child an absolute dependence for every decision, no matter how trivial; and so the child reaches the years of discretion unable to meet the ordinary stresses of life. Such children remain infantile and their subsequent existence becomes a series of desperate attempts to escape realities. Mother-love in its highest ecstasy can blight or even crush the human soul with a cruelty akin to that of the Inquisition. And the irony of it is that this is all done in blissful innocence and with intentions furthest re-

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moved from those of hate. These mothers are not necessarily ignorant; many are well educated and some even consider themselves cultured. The state correctly demands licenses from those who are responsible for human lives, but it makes no provisions for the privilege of rearing children. One wonders whether such enlightenment will be attained in the future state. Bearing children and rearing them may some day be regarded as entirely separate callings. In recent decades due to economic pressure small families have become necessary, which probably accounts in some degree for the increase in the number of highly sensitized individuals, for it is obvious that the smaller the number of progeny, the greater the hazards of maternal domination. When a child is a single one, the chances of sensitization are almost certain. A single child starts life with almost insuperable handicaps.

Fathers are less responsible; first, because they have no nutriment-giving breasts; secondly, because they see infinitely less of their children than the mother; and thirdly, because only too often the mother is jealous of paternal influence. If a child could select the best atmosphere in which to be sensitized it would be a household with a fussy and dominating mother, a father whose interests are in everything except the rearing of children, and a horizon in which no or few rivals are visible, especially of his or her own sex.

Relatives, other than parents, have their scope for harm but only when they substitute as parents and the victim is still within the impressionable age.

There are other protective mechanisms. One of the characteristics of advancing civilization is the invention of means to soften the edges of the tools of living and the "bludgeonings of chance". Consider our subtler and swifter methods of communication, the expansion of industrialism, the enormous refinements in comfort, the vast conquest of the forces of nature.

The slow disappearance of the frontier, which Truslow Adams regards as so important in checking our economic development, has at the same time checked the pioneer spirit, the lust of adventure and aggression that was so marked a trait in the older generations.

Our own science, medicine, has also become a sensitizing influence; for the alleviation of pain, and the refinements of the sick room, while they help us to conquer, at the same time make invalidism much easier and the individual softer. Maladies that were formerly regarded by the public as minor have become major, and it pains one to say that we have developed a breed within our own calling who encourage this distorted perspective.

Certain races are more sensitive than others, but, I believe this is the result of extraneous factors and not of genetic qualities. In general, Southern races are more sensitive than Northern; this is probably due to climate which makes the emotions of Southern people more labile. But there are other reasons that apply to isolated cases. Our Southern women are notoriously more sensitive because of the chivalrous attitude of their male associates. Primitive peoples are less sensitive than those that are civilized, and in a general way, the greater the civilization the greater the potentiality for sensitization.

This is readily understood when one considers that increasing complexity of life brings corresponding mechanisms for protection. It is a remarkable phenomenon that as soon as a primitive folk comes into prolonged contact with a civilized one, evidences of sensitization become manifest. This is well exemplified in our Negroes. Formerly Graves' syndrome was a rare disease in Negroes; at present it is common and appears to be increasing. The notorious sensitivity of the Jews is undoubtedly due to generations of persecution and the development of keen protective mechanisms, as the result of the conflicts engendered by the Diaspora. Religion may be a sensitizing agent only when it teaches irreconcilability in the face of death; if reward is promised, such as heaven or immortality, death arouses little fear. Religion may be regarded as one of the escapes from a world of reality and probably the most widespread neurosis that has affected mankind. Religion is therefore a result and not a cause of sensitivity to life.

Education is the most potent antagonist of neurosis, for fear is bred of ignorance. Scientists are not so subject to anxiety and kindred neuroses as those who lack the scientific discipline. Scientific discipline connotes the ability to doubt while the sensitive depend for their conduct in life upon dogma. It is not in the diffusion of knowledge that education fails, but rather when it teaches such protective mechanisms as the inviolability of tradition, rigidity of thought, the sanctity of conventions, and the virtues of a soft, smug, and complacent life.

Thus far, in this discussion, the influences that make for sensitivity, either of the individual or of the mass, have been entirely environmental. Sensitivity, in other words, represents a mutual reaction, a give and take between the ego and its surroundings that is progressive as life increases in complexity and intensity. I do not believe that sensitivity is congenital except under two conditions. First, when there is a congenital deficiency in the individual's physical frame or the basic intellectual potentials, so that he masks his deficiency in various forms of compensatory mechanisms. Lord Byron with his deformed foot is an example of physical inferiority that comes readily to mind. Acquired deformities, especially if they arise in the early years, may result similarly. The second is the sensitivity both to phys-

ical and mental insults that accompanies that curious morbid state known as status thymicolymphaticus which has all the earmarks of a congenital lesion. It is well recognized that this lesion is a common background upon which Graves' syndrome is engrafted. Indeed, over 95 per cent of the fatal cases of this malady are associated with status thymicolymphaticus. The cause of the sensitivity is a mystery.

The manifestations of a heightened sensitivity to life are largely functional and range from the milder aberrations of behavior to major hysteria and even to manic depressive psychoses. I have already referred to certain maladies of organic character such as Graves' syndrome, hypertensive disease (essential) and peptic ulcer, which I believe are of psychogenic origin. Of these, Graves' syndrome is the most exquisite example. I tried to show in a previous study[1] that this syndrome expresses biologically a series of events ranging from the primitive form variously represented under the terms, autonomic imbalance, Basedowid, pre-Basedow, formes frustês to the complete florid stage conventionally termed exophthalmic goitre. The dominant characteristic of this malady is a constitution that is largely phenotypic. The disease occurs in highly sensitive, emotionally unstable folk and the clinical manifestations are brought to light as a general rule either by catastrophic emotional insult, or by less perceptible reiterated mental shocks. The hyperthyroidism is secondary, probably the most important clinical manifestation, and therefore should by no means be considered as synonymous with the disease. Psychotherapy is an important adjuvant in the treatment of the disease, not only before but even after thyroidectomy. The evidences of sensitivity in hypertensive disease (essential) and in gastric ulcer are not so apparent as in Graves' syndrome, but inasmuch as conflict enters so largely into their genesis, the matter of sensitivity is entirely a relative one.

It is agreed that neuroses represent an attempt at adjustment, an escape from the world of reality and an attempt to hurdle life's difficulties. As Wechsler aptly puts it, a neurosis is the penalty for the unsuccessful attempt to grow up. Such escapes manifest themselves in a variety of disturbances of conduct, the phobias, anxiety neuroses, exaggerated narcism, compulsion neuroses, hypochondriasis, doubting follies, psychasthenias, etc. But there is a host of other manifestations of escape that are not as a rule classed among the neuroses; these aberrations of behavior are so slight that society not only condones but even approves of them.

One of the most conspicuous symptoms of sensitivity is the overdevelopment of the instincts and the senses. This is obvious when one considers that an escape from the realities of the environment leads naturally to a greater dependence upon one's own inner fires. And thus the instinct of self-preservation becomes extraordinarily refined and enhanced; jealousy, hate, love, and fear become powerful weapons. The emotional life of those who are thus affected covers a wide range. They lack poise and phlegm, they become profoundly narcisistic, self-absorbed and self-pitying. Sensuality becomes their predominant characteristic. They take easily to drink and sedative drugs because they want to forget. I take it that this represents a sort of death wish. Their passions are always aflame. They indulge in sexual excess. If these are for various reasons frustrated, perversions and especially homosexuality often ensue. As a consequence their married lives become unstable. They plunge deeply into religious cults and into philosophies tinged with mysticism. They pass speedily from experience to experience and become a restless, wayward, Bohemian folk. From this group come most of our artists, poets, writers, musicians, painters and actors. We need them badly; indeed, far more than most of our captains of industry, for it is they who bring romance into our lives. It is the idea of romance that people associate with Southern climes. That is why Cohen-Portheim says, "The North craves the South. The South satisfies feeling. The cooler regions of the earth lead to a scientific outlook on life, but it leaves an emotional void. The Northern lands have many admirers but few lovers."

Hovering over the lives of the abnormally sensitive is the constant menace of self-destruction. The vast majority of suicides, especially the young, come from these people. It may seem strange that one who loves a sensuous life so intensely should seek to destroy himself; but the gratification of the senses becomes so constant and so intense, that living becomes a long-drawn-out pain and it requires but a push (and it is often a ludicrously trivial one) to make a decision between life and death. Security is attained at last, but in the grave.

Having made this indictment, what is the outlook and what is the remedy? The outlook is not rosy, so far as the immediate future is concerned. We cannot stay the hand of material progress nor, indeed, would we be willing to do so. Nor is the permanent solution of the economic problem and in consequence the stabilization of the social order an immediate prospect, so that social service seems likely to continue as a vital public and private function. The simple life, except in the sense of a change of values must, therefore, remain an enticing dream. And so if it is difficult to mould our environment the solution must come from within

ourselves. I have already hinted at the remedy in the foregoing remarks. Escapes are not always avoidable in what Powys calls "our conscious life between two incredible eternities", but if escape we must the only legitimate and reasonable one is knowledge. By knowledge, one does not necessarily mean culture, although it helps by affording a finer sense of values, but the study of man himself. Although we cannot select our mothers and other relations, at least we can teach them. We can teach them and the rest of humanity the dark menaces of overprotection. We can teach them to face realities squarely, to appreciate the drifts of civilization and to cultivate self-reliance and self-determination and thus to achieve maturity. To accomplish this they must be taught the workings of the human mind, the springs of human motives, the proper control of the instincts and senses. Spiritual values will be prized above the material. Money will be regarded in the light of a means and not an end.

The beginnings of such an education are already visible in the recent diffusion and popularization of knowledge in regard to child and adult hygiene. But it must go much farther. The responsibility of the medical profession for this knowledge and the part that it must play in it is very important. I fear we have been rather remiss in trying to maintain the traditional medieval isolation of medical lore. The practice of medicine is no longer a matter of magic or mystery, however dark its workings may be. One of the crying defects of practice of medicine today is the appalling lack of public knowledge of the principles of medicine, not only the functional but organic. There is need to recognize not merely the external conflict with disease but the disease caused by the internal conflict. Doctors must teach people how to live as well as how not to die.

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VERMONT STATE MEDICAL SOCIETY

ATROPINE FEVER IN EARLY INFANCY*

BY C. K. JOHNSON, M.D.†

ALTHOUGH I am aware that there have been several papers written on this subject within recent years, I feel sure that many of you are not familiar with the fact that atropine may cause a rise of temperature, at times very high, in a few susceptible infants. This drug is now quite generally used in young infants in the treatment of pyloric spasm or for gastro-enterospasm (colic).

It is usually used in solution made with one grain of atropine in two ounces of distilled water. One drop of this solution would represent approximately 1/1000 of a grain. The usual dose is one drop in a teaspoonful of water a little before each feeding and this dose slowly increased to two or three drops at each feeding if needed to control symptoms.

White states that atropine will cure the majority of infants with functional vagogenic gastro-enterospasm and given the principal symptoms of this condition as "(a) Extreme fretfulness, aggravated by feeding, with inability to nurse because of cramps or (colic) with (b) occasional projectile vomiting, plus diarrhea or constipation, with or without visible gastric or intestinal peristalsis and (c) without the olive shaped tumor of organic pyloric stenosis."

This tendency to spasm is most frequently found in infants under two months of age. The cause of the fever seems somewhat uncertain. Benzing believes that the fever is of central origin, the atropine circulating in the blood is a parasympathetic toxin attacking the heat regulating vegetative centers, then, owing to the paralysis of the heat inhibiting centers, heat formation is excessive.

Physiological effects of the atropine show a scarlet flush of the face and body, dilated pupils, shallow and more rapid respiration and increased pulse rate.

Howell doubted the existence of any one center, considering that there were several coördinating systems involved. Atropine diminishes glandular activity, especially of the sweat glands and the absence of perspiration may be at least in part responsible for the fever. Howell cites Zunt's case of a man without sweat glands who developed a high fever on making the slightest effort in warm weather and he further states that this is analogous to the struggling infant deprived of its ability to perspire by atropine, but by this analogy all infants given atropine should develop a fever. It is, however, estimated that less than ten per cent of them do so.

Wood[1] attributes the temperature to paralysis of the thermogenic centers in the spinal cord, and the final fall to vasomotor paralysis.

Solis-Cohen[2] believes that atropine fever re-

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sults from increased heat production due to direct stimulation of the heat center.

Benzing[3] in a study of fifty infants distinguished two groups in regard to type of reaction to atropine. The first group were definitely hypersensitive to atropine. In this hypersensitive group he found infants on a concentrated diet with insufficient fluid and thinks that dehydration favors the fever. A marked increase of tolerance was noted when the dose of atropine was very slowly increased.

Dresel states that there is much experimental evidence to prove that the parasympathetic fibers inhibit heat, and that the sympathetic fibers stimulate heat production and that atropine is a parasympathetic inhibiting agent.

Alarcon[4] stresses that individual susceptibility varies, some infants showing signs of intolerance after one drop of a 1-10,000 solution, and cites another infant who by mistake received twenty drops with each feeding, without untoward effect. He states that this phenomenon may be explained as follows: it is due to the pharmacodynamic action of the minute doses of atropine which excite the vagosympathetic system, stimulating the secretory function of the glands, chiefly of the thyroid, the resultant transient hyperthyroidism, by accelerating metabolism, produces abnormal heat. The cutaneous hyperemia and suppression of sweat which accompany the fever are vagal phenomena parallel with or consequent to it; they cannot, however, be considered as causes of the rise of temperature, although they are contributing factors. He states further that an important factor in the production of fever is the thermolability of the tiny organism due to innate vagotonia, together with its transitory inability to control and direct its vegetative physiology by means of the skin.

McNab reports a case, that of a four year old child who died after eating belladonna berries, the temperature reaching 110 degrees F., a few hours before death.

Beehler[5] has reported the case of a female twenty-five years of age who took three grains of atropine. She developed an axillary temperature of 105 degrees F. and a red rash. Atropine was found in the urine four hours after ingestion and one drop of the urine in a cat's eye dilated the pupil in five minutes.

Benzing and others report that infants with atropine fever show a neutrophilic leucocytosis.

This atropine fever is characterized by a sudden onset and may last a few days or may disappear rapidly without any aftereffects. Stopping the drug causes the temperature to disappear, only to reappear, when more atropine is given.

Marriott[6] says that atropine may at times lead to an increase of body temperature to 102 to 104 degrees F., not accounted for by any infection. He also states that atropine fever is not necessarily a contraindication to its use, although it is usually well to diminish the dose somewhat.

Neff[7] states that not infrequently small doses of atropine cause flushing of the skin but that this is not dangerous or injurious. Several other textbooks on pediatrics have been consulted, some of them just off the press, and about fifty per cent of them do not mention fever in conjunction with atropine therapy.

Munns[8] reports a case of atropine fever reaching 109 degrees F. with recovery. He states that the reaction occurs only in very young infants and may occur when an ordinary dose is given. He further states that the fever is controlled by hydrotherapy and that there are no harmful aftereffects. He does not attempt to explain the phenomenon but considers the drug a safe remedy if the dose is increased with caution.

Bass[9] was one of the first to describe that which he called "the hypertonic infant" and recommended the use of atropine in these cases.

Dennett[10] describes the hypertonic infant and strongly recommends the use of atropine. He speaks of atropine as a specific and that its use in vomiting is of diagnostic value. He also calls attention to the fact that this drug may at times cause hyperpyrexia.

It is this fact, that many textbooks do not mention the possibility of atropine causing a temperature at times very high, and the fact that atropine is now quite frequently used in the treatment of these small infants that led me to present this paper.

I have used atropine quite extensively in infant therapy for some years and during this time have occasionally seen infants that were rather sensitive to this drug. I have, however, not seen many who developed high fever.

I wish to cite but two cases of atropine fever at this time.

CASE 1. A. H., born December 28, 1933, weight nine pounds and three ounces, birth normal, breast with complemental feeding from the first day. On January 1, the four day old infant vomited a little and on the sixth day the vomiting had increased and was thought to be projectile in character so at 6 P.M. 1/500 grain of atropine was given by mouth, this dose being repeated at 10 P.M., also at 6 A.M. and at noon the following day. I saw this infant at 6 P.M. on this day because of a rectal temperature of 103.3 degrees F. The respiration and pulse were rapid and there was a red flush over the entire body. The physical examination was otherwise negative. The atropine was stopped and fluids given, with a drop of temperature to 99.4 degrees F. at 10 P.M. There was no further rise of temperature and no more vomiting of account. It has been stated that this hyperthermia caused by atropine had a beneficial effect on the vomiting in some cases, as might have been the case in this instance. However, one would hardly wish to induce the high fever purposely. I feel that, in many of these in-

fants three or four days old that begin to vomit, the cause is often a poorly adjusted formula rather than any instability of the infants' nervous system.

CASE 2. C. S., born January 23, 1933, weight seven pounds and two ounces. On February 18 when twenty-five days of age and a weight of six pounds and ten ounces, with some dehydration the vomiting which had been mild before became more marked and at 6 A.M. temperature was 99 degrees F. rectal. At 7:45 A.M. 1/1000 grain atropine was given, at 9:30 the temperature was 102 degrees F., at 10:30 A.M. 1/1000 grains atropine, and the same at noon, the temperature then 101.4. At 2:45 1/1000 grain of atropine was given and the temperature at 3 P.M. was 107 degrees F. rectal. I first saw this infant at 5 P.M. No more atropine was given and the rectal temperature was 100 at 10 P.M. and remained down.

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MISCELLANY

ANNUAL MEETING

The Annual Meeting of the Vermont State Medical Society will be held at Rutland, Vermont, October 17 and 18. The following provisional program is submitted:

1. President's Address. George G. Marshall, M.D., Rutland.
2. Paper. Augustus B. Wadsworth, M.D., Director of Division of Laboratories and Research of the State of New York, Albany, New York.
   Topic: "Practical Limitations of Vaccine and Serum Therapy."
3. Paper. Ward Woolner, M.D., Past President of the Ontario Medical Association; Past President of the Medical Health Officers Association, Ayr, Ontario.
   Topic: "Rural Health Problems, the Problems Themselves and Their Control."
4. Paper. C. F. Whitney, M.D., Professor of Physiological Chemistry and Toxicology, University of Vermont, Burlington, Vermont.
   Topic: "Chemistry in Relation to the Practice of Medicine."
5. Paper. N. D. Smith, M.D., Mayo Clinic, Rochester, Minn.
   Topic: "Obscure Abscesses a Cause of Acute Anal Pain." (Their Diagnosis and Treatment.)
6. Paper. E. J. Rogers, M.D., and L. A. Rabinowitz, M.D., Vermont Sanatorium, Pittsford, Vermont.
   Topic: "Chronic Cough."
7. Paper. Henry A. Christian, M.D., Physician-in-Chief, Peter Bent Brigham Hospital; Hersey Professor of the Theory and Practice of Physic, Harvard University, Boston, Massachusetts.
   Topic: "Types of Edema and Their Treatment."
8. Paper. Foster Kennedy, M.D., New York City.
   Topic: "The Biopsychic Approach to Diseases of the Mind: Its Dependence on Neurology and General Medicine."
9. Paper. O. N. Eastman, M.D., Associate Professor of Obstetrics, University of Vermont, Burlington.
   Topic: "Hysteroptosis."
10. Address. James S. McLester, M.D., President of the American Medical Association, Birmingham, Alabama.
11. Vice President's Address. Frank C. Phelps, M.D., Attending Physician, Porter Hospital, Middlebury; Attending Physician, Vermont State Industrial School, Vergennes, Vermont.
12. Paper. Arthur J. Bedell, M.D., Albany, New York.
    Topic: "Ophthalmological Diagnoses."

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21391

PRESENTATION OF CASE

A forty-four year old Italian woolen worker entered complaining of diarrhea.

During the past three years the patient had had frequent abdominal pains across the epigastrium, occasionally extending in an irregular manner to both lower quadrants. These pains were sometimes present for about a month or more at a time and were also absent for a similar length of time. Four weeks before admission, about an hour and a half after supper, he vomited. He continued to vomit that evening, three or four times in all. At the same time he developed rather severe diarrhea, moving his bowels every one or two hours. The next morning the vomiting stopped but the diarrhea continued. From that time until admission he had about eight bowel movements every day. The stools were always soft and watery, but as far as the patient knew had always been free from fresh or old blood. Accompanying these symptoms he also had a feeling of abdominal distention immediately after eating. Two hours after eating he felt a "pinching, stabbing" epigastric pain which was relieved by food. At times he had a "rolling sensation" and a "rumbling" running down the left side of his abdomen to just below the umbilicus and then crossing to the right lower quadrant, where it stopped. Shortly after meals he had the sensation of "something hard and big" passing down the bowel and getting stuck in the region of the right lower quadrant. His desire for food remained good but the fear of precipitating abdominal distress prevented him from satisfying this desire so that he had practically abstained from eating meat during the past four weeks. During this same period he had cramplike pains in the calves of his legs which had increased in severity. He became progressively weaker and lost about twenty pounds in weight.

The family, marital and past histories are non-contributory. There was no history of tuberculosis or carcinoma. He had had pneumonia twenty years before admission.

Physical examination showed a fairly well-developed and rather poorly nourished man in no immediate distress. The mucous membranes were pale. The heart and lungs were negative. The blood pressure was 104/60. There was slight tenderness in the right lower quadrant. No masses could be felt. There were a few small external hemorrhoids. Rectal examination, however, was negative except for slight tenderness.

The temperature was 98.4°, the pulse 78. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 3,120,000, with a hemoglobin of 50 per cent. The white cell count was 9,700, 66 per cent polymorphonuclears. The stools were small, soft and brown. Three out of seven stool examinations showed positive guaiac tests. No amebae could be found. A Hinton test was negative.

A barium enema showed a definite delay in the passage of the barium at the rectosigmoid junction but then a small quantity of barium entered the descending colon and flowed to the region of the splenic flexure. The remainder of the colon could not be filled but a definite lesion could not be demonstrated.

On the fourth day a red non-ulcerating polypoid mass on the right wall of the region of the rectosigmoid was removed during proctoscopy. The pathologic diagnosis was adenomatous polyp. One week after admission he had considerable vomiting, abdominal cramps and diarrhea so severe as to amount to incontinence. He became severely dehydrated. The blood pressure was 70/50. He was given 4 liters of 10 per cent glucose with marked benefit. One observer at this time felt a small, hard mass in the region of the cecum. Another enema on the tenth day showed that the sigmoid was somewhat spastic but no lesion could be made out. The cecum was quite irregular but a definite diagnosis could not be made. During the examination the patient vomited several times and was unable to retain all of the enema. Two weeks after admission an exploratory laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. HENRY H. FAXON: Intermittent epigastric symptoms of three years' duration would immediately suggest a gastric ulcer until proved otherwise. Radiation from the epigastrium to both lower quadrants, however, is distinctly atypical for any common type of pathology and raises doubt as to how much weight to lay on this part of the history.

"Four weeks before admission, about an hour and a half after supper, he vomited." There is such an abrupt transition between the rather mild three year story and the severe symptoms which have been present for only four weeks that one is forced to raise the question whether this is a new lesion or some type of fairly dra-

matic change in that of which he originally complained.

"Two hours after eating he felt a 'pinching, stabbing' epigastric pain which was relieved by food." Relief so long after eating would be rather atypical of an ulcer and might better fit the picture of gastric malignancy.

The "rolling sensations" and rumblings spoken of are suggestive of a certain amount of obstruction with which the vomiting would also be in keeping. However, if the obstruction (which with his diarrhea would have to be incomplete in nature) were located in his upper intestinal tract it should not have radiated to his lower abdomen. If, on the other hand, as seems more likely, the site of the obstruction were in his large bowel, it is difficult to correlate the epigastric pain of which he also complained.

"Shortly after meals he had the sensation of 'something hard and big' passing down the bowel and getting stuck in the region of the right lower quadrant." Again in all probability this is gas in the face of a certain amount of obstruction.

In an Italian the possibility of some parasitic infection as a cause for the whole picture cannot be overlooked and with the muscle pain mentioned here trichiniasis comes to mind. There is no report subsequently, however, of any increase in the eosinophilic count which would be expected in most of these conditions. We find later that he had a considerable degree of anemia, so that the pain in the calves of his legs may have been on the basis of insufficient oxygenation of these muscles.

His weight loss most probably could be largely attributed to his very restricted diet, although of course, the effects of malignancy might also be a factor.

Tenderness in the right lower quadrant is often hard to evaluate since gas in the cecum is always somewhat tender to pressure, and granted the assumption that a certain amount of large bowel obstruction was present I would expect that there would be more than the average amount of gas in his ascending bowel.

His secondary anemia, it would seem to me, could best be explained in the light of the whole picture as being due to malignancy. Taken in conjunction with the normal temperature it would seem that his pathology was not primarily infectious in type.

"Three out of seven stool examinations showed positive guaiac tests." Certainly this clinical finding is of more significance than his story that blood in the stools had never been noted. However, these two factors taken together would suggest that the degree of hemorrhage at any one time had never been severe.

"No amebae could be found." If properly carried out this should rule out amebic dysentery as a cause of his symptoms.

Although no definite diagnosis was made on the first x-ray examination, this study gives more of a lead than anything we have met so far. Certainly from this report it can be definitely stated that some abnormality involving the large bowel was present.

"On the fourth day a red non-ulcerating polypoid mass on the right wall of the region of the rectosigmoid was removed during proctoscopy. The pathologic diagnosis was adenomatous polyp." Had any definite abnormality of the mucous membrane of the rectum itself been noted I assume that it would have been stated in this report. The finding of the polyp in itself is of significance chiefly due to the fact that it is well recognized that polyps are apt to be multiple, and it is more than likely that elsewhere in the large bowel additional polyps might be found any of which conceivably could have undergone malignant changes.

"One week after admission he had considerable vomiting, abdominal cramps and diarrhea so severe as to amount to incontinence." I cannot stress too strongly the added help that it would be to know whether the abdominal cramps referred to were above or below the umbilicus. If the latter were the case I could discount to a considerable extent his upper intestinal symptoms; whereas if these cramps referred to were above the umbilicus some correlation between his large bowel pathology and upper intestinal tract would have to be made. Considering that vomiting is also a noteworthy symptom I am inclined to believe that the upper intestinal tract is probably involved secondarily by mechanical interference.

"He became severely dehydrated." Certainly with diarrhea of the extent this man had, dehydration would be a prominent symptom if not vigorously combated by a high fluid intake.

"The cecum was quite irregular but a definite diagnosis could not be made." This finding together with the fact that one observer believed he felt a mass raises the question as to whether the lesion might not be in the cecum or ascending colon. Malignancy could well account for the secondary anemia and it is characteristic of right-sided lesions to bleed to a considerable extent. However, with a right-sided lesion a diarrhea of the degree that he showed would, I believe, be unusual, and the obstructive symptoms also, which I cannot but feel play a part in the picture, would be more unusual with carcinoma of the cecum than with a lesion further along the large intestinal tract.

We are not told at any time what x-ray studies of the stomach and small intestines showed. Such an examination would, of course, have been of value from a threefold point of view: It would be of help in excluding any gastric pathology; it would help clear up the problem of upper intestinal obstruction; and it would give the definite lead to any spontaneous anasto-

mosis that might have occurred between a lesion of the large bowel and the stomach or upper abdominal tract.

When the above facts are evaluated the most significant definite points we have to go on are the fact that a forty-four year old Italian has a bleeding lesion of his gastrointestinal tract associated with a diarrhea and with x-ray findings suggestive of a lesion in the large bowel. Many of his complaints, however, are referable to the upper intestinal tract. I believe that he had malignancy of the large bowel, most probably located in the transverse or descending colon. I picture the lesion as causing a partial obstruction of the large bowel and, due to extrinsic involvement, a certain amount of associated upper intestinal obstruction as well.

Clinical Diagnosis

Carcinoma of cecum (colloid adenocarcinoma, grade III).

Dr. Henry H. Faxon's Diagnosis

Carcinoma of large bowel (probably transverse or descending colon).

Anatomic Diagnoses

(Carcinoma of cecum).
Operative wound: Transverse ileocolostomy; resection of cecum.
Peritonitis, acute localized.
Lung abscesses, bilateral.
Bronchopneumonia, left.
Bronchiolitis obliterans, very slight.
Pleuritis, chronic fibrous, bilateral.
Benign adenomatous polyp of rectum.

Pathologic Discussion

Dr. Tracy B. Mallory: The preoperative diagnosis in the house was in agreement with Dr. Faxon—that there was a carcinoma of the large bowel—although they placed it in the cecum rather than in the transverse colon, evidently taking the questionable mass in the right lower quadrant fairly seriously. The patient was transferred to the surgical service where a right rectus incision was made and the peritoneal cavity explored. A rather large adherent tumor mass was found in the ascending colon which extended almost up to the hepatic flexure. The patient's condition was considered too poor to attempt radical extirpation at this time; so an ileocolic anastomosis was performed. Following this operation the patient's condition steadily improved for a two-week period. Then a second operation was performed in the attempt to resect the growth. It was found to be firmly adherent to the lateral wall of the duodenum and it proved necessary to resect a portion of the duodenal wall along with the cecum and ascending colon. The operator felt that very probably a fistula between the colon and duodenum had been present although it was not possible to verify this with certainty from the resected specimen. The pathologic examination of the specimen showed a polypoid, fungating, slightly ulcerated tumor mass, from the base of which induration extended a considerable distance into the adjoining intestinal wall. The microscopic examination showed a colloid adenocarcinoma and also proved that the duodenal wall was deeply invaded.

Following this second operation the patient's course was progressively downhill. Abdominal spasm was noted for three days but then disappeared. He ran a moderate fever ranging from 100° to 101° and the respirations, after remaining at 20 for the first fifteen days, climbed to 30. Coarse râles were heard at the left base and slight dullness was made out. A portable chest plate showed mottled dullness involving the mid portion of the left lung field and the entire right lung field. It was considered characteristic of bronchopneumonia.

The autopsy showed two localized areas of peritonitis — one immediately beneath the abdominal incision, the second occupying the entire cecal bed and extending up to and around the lower pole of the right kidney. The surgical suture lines all appeared tight and in good condition and no evidence of a fistula could be made out in the remaining portion of the duodenum. It was felt that this localized peritonitis was probably not an important factor in the fatal termination. A much more important lesion was a series of lung abscesses varying from 1.5 to 6 centimeters in diameter, which were filled with necrotic, blackish, foul smelling material. The abscesses were apparently still in the developmental stage and had not as yet broken down to the point where significant amounts of their content had been discharged into the bronchi. The adenomatous polyp which had been found on proctoscopy proved to be at the junction of the rectum and sigmoid. Microscopic sections through its base confirmed the previous diagnosis of a benign polyp.

Cases of this type, where a carcinoma of the large bowel is associated with one or many benign adenomatous polyps, are very frequent and present strong presumptive evidence for the development of malignancy from benign polyps. A less common lesion, of which I thought when I read over this history, is the extreme grade of multiple polyposis in which almost every inch of the bowel wall is covered with the polypoid masses. Such cases not infrequently give a history of prolonged intractable diarrhea which may be indistinguishable from the stories of cases of ulcerative colitis.

Dr. Faxon's attempt to correlate the epigastric with the lower abdominal symptoms by the

assumption of a tumor in the transverse colon which had invaded the stomach or small bowel, with or without the establishment of a fistula, was an ingenious solution of the perplexing symptomatology, and the actual finding of a carcinoma of the ascending colon involving the duodenum is so close that he deserves considerable credit for having made it.

CASE 21392

Presentation of Case

A forty-one year old single American teacher entered complaining of fever of two weeks' duration.

Three months before entry the patient had an attack of "ptomaine poisoning," apparently due to lobster, which kept her in bed for two weeks. She was acutely ill during the first week with abdominal pain and fever. After this she was perfectly well until two weeks before entry when she developed a sore throat and "ached all over". She went to bed and stayed there until admission with a temperature of 101° to 102° every afternoon. After about a week the sore throat subsided somewhat, although it was red and covered with membrane. The cervical glands were enlarged. During the first week she sweat a great deal but had no chills. There was no cough or pain in the chest. She had herpes of the lips and nose. During this illness she had been given digitalis. There was no vomiting, diarrhea or abdominal pain. She had no urinary difficulties. Her menstrual history was negative.

Her family history is non-contributory.

She had an unknown fever at the age of five, scarlet fever at twelve, diphtheria at twenty-three and repeated attacks of tonsillitis. At the age of eighteen her appendix was removed. Seven years ago she was said to have a heart murmur. During the past three years she had slight dyspnea and palpitation upon exertion. She was seen by a cardiologist two years before entry and a diagnosis of mitral stenosis was made. She was not reëxamined until four months before entry and the heart condition had not essentially changed.

Physical examination showed a pale, thin, sick-looking woman lying in bed breathing a little rapidly. Her throat and tonsils were moderately infected and dull red but contained no exudate or membrane. The heart was enlarged, the left border of dullness being 11.5 centimeters from the midsternal line, the right 3 centimeters. There was a systolic wave over the precordium and a short presystolic thrill at the apex. The sounds were loud. P_2 was slightly louder than A_2. There was a middiastolic rumble heard best toward the axillary line. The chest showed some dullness with bronchial breathing, decreased breath sounds and a moderate number of moist râles at the left base. The liver was felt 12 centimeters below the costal margin in the midclavicular line and was non-tender. There was no evidence of fluid in the abdomen. The spleen was not felt. There was no edema of the ankles. No petechiae were seen. The knee jerks were not obtained.

The temperature was 102.1°, the pulse 110. The respirations were 38.

Examination of the urine showed a specific gravity of 1.028 with a trace of albumin. The sediment contained 30 white blood cells, 15 red blood cells and numerous bacteria. The blood showed a red cell count of 4,060,000, with a hemoglobin of 70 per cent. The white cell count was 24,000, 87 per cent polymorphonuclears. The stools were negative. An electrocardiogram showed normal rhythm, rate 80, broad notched P_1 and P_2 and right axis deviation. The P-R interval was 0.18 seconds.

Examination on the third day showed no real changes except those in the lungs. The right chest was quite dull and the râles persisted. The previous bronchial breathing had subsided. In the left axilla, however, there was an area of dullness with a suggestion of bronchial breathing and râles. No friction rub was heard. There was very little cough and no sputum. She rapidly went downhill. The temperature, which at first went down to between 100° and 101°, rose on the fourth day to 103.5°. The white cell count rose to 33,200. The edema increased. She became progressively weaker, moderately cyanotic and died on the fourth day.

Differential Diagnosis

Dr. William B. Breed: I do not like to call this intestinal episode ptomaine poisoning if it was simple food poisoning. She was well after two weeks. Well, either it was food poisoning and had nothing to do with the present illness or it was not food poisoning, from lobster at least, and it did have something to do with it. I cannot tell which it was. She was well for two months and I am tempted at the moment to exclude this episode from the present illness, although it may become significant later. We will say that the present illness began two weeks before entry when she developed the sore throat and ached all over. The throat was red and covered with membrane. I should like to know a little more about the membrane, whether it was an exudate which resembled a membrane or a true membrane. I should like to know why she was given digitalis. We may find out later. She had an acute sore throat with cervical adenitis and herpes. We do not know about a culture or what the white count was, and we do not know whether she had taken any other drugs for her sore throat, such as amidopyridin; so we think either of acute streptococcus infection or possibly agranulocytosis. It may be one of the leukemias.

There is probably no diphtheria in spite of the mention of the membrane.

With this story of repeated tonsillitis and the evidence she has of rheumatic heart disease, this is a perfectly good story for acute rheumatic fever. I am not clear about the association of herpes with rheumatic fever. I have never seen it. It may be associated with it or it may be incidental. I still do not bring in the food poisoning. The observation that P_2 is greater than A_2 does not mean anything unless there is an added note that P_2 is accentuated. We know that she has a sore throat and presumably tonsillitis, and there may be a flare-up of rheumatic infection associated with the sore throat. Of course, the fact that she was "aching all over" in the first part of the history means that she was toxic. There is no evidence pointing to joint involvement. However, we know that that is not necessary for a flare-up of a rheumatic nature. The respirations are high. On physical examination there is dullness with bronchial breathing, decreased breath sounds, and a moderate number of râles at the left base, so that she may have some process in the chest, either bronchopneumonia or possibly something of an embolic nature.

The P-R interval would indicate in general that she was not suffering at the time from recurrence of a rheumatic infection, and although that is not an absolute criterion it is a good piece of evidence. The summary does not say anything about a throat culture. She certainly does not have agranulocytosis or leukemia. Was a culture taken?

DR. TRACY B. MALLORY: It is not recorded.

DR. BREED: Presumably this is a streptococcus throat infection, although we do not know. We do not know about a blood culture. I presume that they were taken. She certainly had an overwhelming infection because she died of it. I think that is clear, and I should doubt that she died of rheumatic infection. She had some bronchopneumonia, probably bilateral, but it does not seem reasonable to expect that she would die as suddenly as that from bronchopneumonia, as mild as it appeared to be. I do not know whether we are at a deadlock here because we have no information or because we are so stupid we cannot make a diagnosis, but the evidence that we have points merely to an overwhelming upper respiratory infection.

A PHYSICIAN: How about subacute bacterial endocarditis?

DR. BREED: The course is too rapid even for malignant endocarditis.

A PHYSICIAN: She may have had symptoms for some time before she came in.

DR. BREED: But she was well for two months.

A PHYSICIAN: How about metastatic abscess, then?

DR. BREED: I should not expect it to be found.

A PHYSICIAN: I think her death might be caused by pneumonia but there is a possibility of acute or subacute endocarditis.

DR. BREED: That is merely a guess. All we can do is guess.

I cannot make a definite diagnosis other than chronic rheumatic heart disease with mitral involvement. She died of infection. I do not know what infection other than just plain tonsillitis and bronchopneumonia. She may have had septicemia. I am sorry that the blood culture was not forthcoming.

CLINICAL DIAGNOSES

Acute rheumatic fever.
Pneumonia (rheumatic)?
Rheumatic heart disease with mitral stenosis.
Uremia?

DR. WILLIAM B. BREED'S DIAGNOSES

Chronic rheumatic heart disease with mitral involvement.
Acute infection—unknown etiology.

ANATOMIC DIAGNOSES

(Acute rheumatic fever ?).
Endocarditis, chronic rheumatic, mitral with stenosis, aortic.
Cardiac hypertrophy, rheumatic, slight.
Pulmonary thrombosis.
Pulmonary infarcts, bilateral.
Pulmonary congestion and edema.
Hydrothorax, bilateral.
Ascites.
Petechial hemorrhage of peritoneum, pleura and pericardium.
Pleuritis, chronic.
Cyst of right ovary.
Operative scar: Appendectomy.

PATHOLOGIC DISCUSSION

DR. MALLORY: I was not too sure of the cause of death after the autopsy was over. She had a very definite, fairly severe old mitral stenosis. We could not find any fresh vegetations on the valve. There were, however, two or three minute vegetations, perfectly consistent with acute rheumatic vegetations, on the aortic valve which otherwise showed only very slight thickening. We did not find any sign of Aschoff bodies in the heart muscle. That means almost nothing, because we do not run through more than two or three blocks of heart muscle per autopsy and if you want to find Aschoff nodules you have to run fifty blocks to be certain of getting them. They can only be found with very high incidence provided you make the search for them almost a research job in each case.

The pulmonary complications were peculiar.

A lot of interest has developed in the last two or three years in the question of so-called rheumatic pneumonia and any time a rheumatic case dies with anything in the lungs everybody wants to know whether it is rheumatic pneumonia. I find it very difficult to get any idea what rheumatic pneumonia is. There are many clinical reports in the literature and a few of them have some appended pathologic notes written almost invariably, I should say, by a clinician who has attempted to interpret the microscopic sections. I cannot make head or tail of the descriptions. There is one paper*, based on only two cases, however, that is fairly good from the pathologist's point of view. In most cases there are multiple areas of hemorrhage throughout the lung, rather sharply focal, looking very much like infarcts, but usually not leading to complete necrosis of the lung tissue. You can also find, microscopically, lesions in some of the very small pulmonary arteries in the immediate neighborhood of the hemorrhagic foci. Histologically the lesions are about half way between infarct and pneumonia in appearance. We have seen two cases here in children with acute rheumatic fever that correspond to that picture. We have seen a lot of atypical pneumonias of other kinds in other rheumatics, and so far I have no clear conception as to whether there is a rheumatic pneumonia entity. This woman had a large number of fairly small but definite infarcts scattered throughout the lung. There was no doubt of their being real infarcts. The alveolar walls were becoming necrotic and we could find thrombi or probably adherent emboli in the arteries leading to each of the infarcts. We did not find any thrombi in the right side of the heart, so it is unlikely that they came from there. I imagine that she was getting emboli from some undiscovered phlebitis of the leg. One always thinks of pulmonary infarcts as a surgical or rather post-surgical disease. As a matter of fact pulmonary infarcts are fairly common on the medical wards but are almost impossible to diagnose except in very characteristic cases where you have a localized pleuritis and frankly bloody sputum or a lead in the form of an obvious phlebitis. One certainly can have thrombosis in the deep veins of the leg which is absolutely silent. The postmortem blood culture was negative. There is no proof, therefore, what the fever was but on the whole rheumatic fever seems as good a bet as any, mostly because of the indefiniteness of the findings.

DR. BREED: It is unusual for a person of her age to die as quickly as this of rheumatic fever, although children die of rheumatic fever oftener than is generally supposed.

A PHYSICIAN: How about the liver?

DR. MALLORY: It was normal in size. It weighed 1600 grams which is within normal limits. The spleen weighed 225 grams. She simply had a low diaphragm.

DR. BREED: We all agree that she probably had an old mitral stenosis and that she died of acute infection, with a question of rheumatic fever. I guess that is the best we could say.

*Fraser, A. D. The Aschoff nodule in rheumatic pneumonia. Lancet **I**, 70, 1930.

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TUBERCULOSIS — AN ANOMALY

TUBERCULOSIS holds a unique place among the communicable diseases—unique, that is, in the attitude of the public, of physicians and of public health officials. With other infectious diseases, the first evidence of their presence starts the operation of a series of events carefully planned to prevent their spread. The first act, after initiating treatment of the patient and reporting the disease, is to search for the source of the infection. While this quest is going on, precautions are taken to prevent the patient from passing on the virus to others. The sick one is surrounded by safeguards; those nursing the sick are instructed in ways for shielding themselves and others against the infection; the room or the house may be quarantined or the patient removed to an isolation hospital. In our archaic handling of that disease of low transmissibility, leprosy, we even banish the leper to exile. If the source of the disease is found to be a harborer of diphtheria bacilli, we sequester the carrier until cultures show the disappearance of the bacilli, and, sometimes, when they remain persistently positive, we, if permitted, remove the tonsils. The typhoid carrier is excluded from all occupations connected with food handling, and when we are successful in our persuasion, we take out the gall-bladder.

The yellow flag, the quarantine card, the gossip of neighbors warn that danger is present. But, with tuberculosis, the order is changed. The source, the open case, spreading living tubercle bacilli, is sympathized with, precautionary instructions may or may not be given, and the attending physician, when there is one, is frequently casual in the announcement of his diagnosis. And, as our city and state vital statistics show, he may report the case to the local health authorities sometimes as late as a month or a week before death, and all too often after the demise of the patient.

Our city, county, state and veterans' hospitals are intended for the removal of the infectious patient from his or her surroundings, but frequently the waiting lists leave the open case at home. But many of these institutions prefer the favorable case. For chronic phthisis sufferers there are hospitals especially planned for their care, but there are too few of them, and it is often impossible to persuade such a patient to leave his home and family circle and to enter an institution known as a resort for incurables. So the source of this disease may be allowed to go about his occupation as long as he is able and then to rest and die, likely, to spread infection in the midst of his susceptible relations and friends.

Tuberculosis, we realize, does not present the immediate and frightening menace of diphtheria, scarlet fever or smallpox, but it causes a greater economic loss, a heavier drain on the efforts and funds of our charitable organizations and a greater sacrifice of human lives.

The voluntary, philanthropic societies have been wholly admirable in their efforts to control this disease. Their education of the public in the ways of good living, of the predisposing causes and early symptoms of tuberculosis; their preventoria and sanatoria for the threatened or afflicted; their vocational training for the convalescent, deserve the highest praise for their nobility of purpose and their splendid achievement. Their aims are both preventive and palliative, but many an active case escapes their search, and having no official authority they can only control these human sources of infection by suasion.

If we examine their programs or those of health departments, we are impressed by their emphasis on the strengthening of health, on early diagnosis, and on the medical and surgical care of the tuberculous. If we read the jour-

nals devoted to this disease, we learn about the biology of the tubercle bacillus, about epidemiological features, new diagnostic aids, the use of this or that remedial agent, the management of hospitals and sanatoria, but in all these publications we find so little about the reporting and control of the open case.

In our private and public campaigns we are now experiencing the operation of the law of diminishing returns. The declining curves of morbidity and mortality from tuberculosis are flattening, and it requires still greater effort to reduce the rate by a point.

Every case of tuberculosis comes from another case, either in a cow or man. Our official federal and state agriculturists are persistent in their determination to rid the country of infected cattle, but we, perhaps because of the difficulties presented by sentimentalism, must be more gentle in the disposition of our human wells of infection. For the control of other communicable diseases our laws are largely mandatory, for the control of tuberculosis they are mostly permissive.

So many of the baffling problems presented by other plagues have been solved, that we are confident of ultimate success in controlling this particular disease. More intensive search and closer control of the open case would speed us along the way. It is, after all, the tubercle bacillus that causes tuberculosis, and it is the purpose of this editorial to focus closer attention upon this neglected, difficult and eminently important feature of tuberculosis eradication in man.

A PHYSICIAN'S HANDBOOK

THE actual practice of medicine proves to be so much greater than and so different from those aspects of practice to which the attention of medical students was formerly directed in order that they might gain a knowledge of disease, that the study of so-called disease entities has been supplemented by more or less complete study of the conditions in which disease arises and develops and must be treated. The supplementary aspects of disease may be characterized as involving environmental factors, among which are the social elements. There is involved, then, first the study of the individual who is sick, and then the study of the society of which he is a part, in which he is sick, on which he acts and which reacts to him.

Several years ago there was published under the auspices of the Massachusetts General Hospital a "Physician's Handbook of Community Resources in Metropolitan Boston, together with Certain Legal Responsibilities", which was made possible by the benefaction of an anonymous donor. The idea of such a handbook seemed excellent, the booklet has filled a long-felt need and the results have justified the undertaking. Has the time not come for a greater development into a larger field of usefulness?

While the legal responsibilities of the physician change little throughout the Commonwealth, the Community Resources vary greatly in the different communities. A physician entering upon practice in a town or city often has little knowledge of the resources of the community and as such a physician is generally beginning practice, he may have had no experience with community resources anywhere. He knows little, therefore, of what his community can and does offer.

Medical students who have been fortunate enough to have intimate contact with the well-organized medical social service of a hospital, may have some conception of what to look for and of how to go about finding what the patient needs, but too often it is only after a blundering and time-consuming experience that the physician learns what his community actually supplies.

The Massachusetts Medical Society, on admitting a physician to membership, gives him a copy of its Constitution and By-Laws. It would not be impracticable and it might be most helpful if each district of the Society provided for its members a handbook similar to that which has proved so useful in the metropolitan area. In addition it would be a step in the right direction if the Board of Registration provided each registrant, at the time of licensure, with a copy of the medical practice act and of other statutes of the Commonwealth pertaining to the practice of medicine. Dealing with the violation of these statutes is part of the duty of the Board of Registration in Medicine and it is not asking too much of the Commonwealth that it provide each registrant with a copy of the law under which he is to practice.

In any case, the issuing by the Society to its new members of a Handbook would be an extension of its educational program into a field hitherto neglected, and an additional recognition of its educational obligation toward all its members.

AN UNFAIR BURDEN

AN analysis of the burden falling on doctors and hospitals is set forth by M. J. O'Malley in the *New York Times* of August 11, 1935. He affirms that several years ago the unpaid hospital bills due to highway accidents had amounted to over $6,000,000. "Hospital Management" in October 1931 estimated that the losses to hospitals in this country incurred in caring for automobile accidents was $5,000,000 a year.

The Research Division of the New Jersey Department of Institutions and Agencies conducted an investigation of highway accidents which dis-

closed that in 1930 the number of persons requiring bed service in hospitals was in excess of 13,000. Nineteen general hospitals were studied as the basis of this estimate. They treated 1781 highway accident patients, who spent a total of 22,441 days in the hospitals. The bills rendered for these cases totaled more than $100,000, but only 56 per cent of this sum was collected.

In 1930 the Ohio Hospital Association found that, of more than $810,000 hospital charges for automobile accident patients, about 50 per cent was considered uncollectible.

During 1931-1933 inclusive, accidents within the metropolitan area of Richmond, Va. brought 4056 patients to the Medical College of Virginia Hospital and over 7000 were given out-patient treatment. The total cost to the community for the three years was $217,000. In Philadelphia, a committee found that charges amounting to $16,410 for patients hospitalized by reason of highway accidents were made with one third unpaid at the close of the year following the accidents. In all cases, fatal and otherwise, studied by this committee, one bill in sixteen was paid.

In England it is provided that the driver of a car involved in an accident causing personal injury must pay for emergency treatment for each patient. Some of our States have tried to have laws enacted which will protect doctors and hospitals in caring for victims of automobile accidents. Senator Miles tried to have the law passed in Massachusetts with this object in view. Lawyers claimed that the bill as drafted was unconstitutional. It is strange that a great injustice cannot be remedied. No one, so far as reports are available, has computed the unpaid bills for doctors' services for these accident cases.

If this burden on hospitals and doctors cannot be remedied by existing or proposed laws, there should be a further study of the subject. It is reasonable to expect that the public will endorse an appropriate remedy.

POSTGRADUATE TRAINING IN MEDICINE

A DISPATCH to the *New York Herald Tribune* of August 15, 1935, quoting from the *German Doctors' Journal* sets forth that the "Aryan" physician of the Third Reich must take a compulsory training course of three weeks once every five years. The evident purpose of this requirement is to elevate the standards of medical practice in Germany.

General practitioners of cities and towns of less than 100,000 residents will be called first to take the prescribed courses with certain exceptions.

This plan apparently recognizes the general belief that doctors as a class do not keep in step with medical progress.

This state of affairs is not confined to Germany and its recognition in the United States is shown by the facilities for postgraduate study which are available for every doctor who is desirous of keeping up to date. The trouble with us is that those who need instruction and training most, are least apt to avail themselves of the opportunities provided by societies and medical schools, with the result that there is a substratum of unprogressive doctors. It has been said that only about forty per cent of our doctors attend medical meetings conducted by District Societies and there are many who do not read medical journals.

Nothing but compulsion will drive such men to study.

If the state confers the right to practice, why should the indifferent doctor be left to practice out-of-date medicine? Just because he has passed the examinations of the State Board, there is no guarantee of his progress in medical education in subsequent years. The government does not let its military surgeons retrograde or even remain static, but requires evidence of improvement at regular intervals. The custom of certification of the doctor's qualifications by one examination is hardly an adequate assurance of his capacity in the competitive field of medical practice.

The German plan is highly commendable, but would meet obstruction in America, in all probability. Our people want freedom from supervision even in the practice of medicine.

A SUIT AT LAW AGAINST AN OFFICER OF THE MASSACHUSETTS MEDICAL SOCIETY

DR. DAVID CHEEVER, Chairman of the Committee on Ethics and Discipline of the Massachusetts Medical Society, has been sued by Dr. Nicandro F. DeCesare for libel. The claimant places fifty thousand dollars as the financial damage. Dr. DeCesare resides at Methuen and has an office at 57 Jackson Street, Lawrence.

This action of Dr. DeCesare is based on statements made by Dr. Cheever in the performance of his duty as defined in the By-Laws of the Society in the investigation of certain charges concerning Dr. DeCesare made to the committee of which Dr. Cheever is Chairman. It became Dr. Cheever's further duty to present the facts bearing on the charges which had come to the attention of his Committee to a Board of Trial appointed by the then President, Dr. W. H. Robey.

It is also of interest to Fellows of the Massachusetts Medical Society that Dr. Daniel Fiske Jones, who originally brought the matter in issue to the attention of the Committee on Ethics and

Discipline, has also been sued by Dr. DeCesare for fifty thousand dollars.

After hearing the evidence, the Board of Trial unanimously reported to the then Secretary of the Massachusetts Medical Society, Dr. W. L. Burrage, its opinion that Dr. DeCesare was guilty of the acts defined in the complaint but after deliberation appended to the report differences of opinion as to the appropriate disciplinary measures to be adopted by the Society.

This report of the Board of Trial was submitted to the Annual Meeting of the Society June, 1935. The Society then referred the material which had been submitted to the Board of Trial to a large committee representing all sections of the Society for a review of the proceedings and a report to be submitted at the next annual meeting of the Society.

It is felt that the Fellows of the Society are entitled to this general statement of this case, but the *Journal* refrains from the publication of details and explanations until the Society shall have taken final action.

The reference of this controversy to the courts also requires a conservative attitude by this *Journal*.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CHRISTIAN, HENRY A. A.M., LL.D., Sc.D. (Hon.), M.D. Johns Hopkins University Medical School 1900. Hersey Professor of the Theory and Practice of Physic, Harvard University Medical School. Physician-in-Chief, Peter Bent Brigham Hospital, Boston. His subject is "Expected Lags Between Clinical Diagnosis and X-Ray Confirmation." Page 593. Address: Peter Bent Brigham Hospital, Boston.

WHITAKER, LESTER R. M.D. Harvard University Medical School 1923. F.A.C.S. First Assistant Visiting Surgeon, Massachusetts Memorial Hospitals, Boston. Associate Member, Evans Memorial for Clinical Research and Preventive Medicine. Instructor in Surgery, Boston University School of Medicine. His subject is "Electrosurgical Cholecystectomy. I. Experimental Observations." Page 596. Address: 41 Bay State Road, Boston.

COHEN, LOUIS. A.B., M.D. Tufts College Medical School 1932. Formerly Rotating interne, Lynn Hospital, Lynn, Mass. Surgical Interne VI Surgical Service, Boston City Hospital, Boston. Now: Interne, Orthopedic Service, State University of Iowa Hospitals, Iowa City, Iowa. His subject is "Anterior Poliomyelitis with Reference to the Occurrence of Two Attacks in the Same Individual." Page 601. Address: Children's Hospital, Iowa City, Iowa.

DAY, HILBERT F. Ph.B., M.D. Harvard University Medical School 1905. F.A.C.S. Professor of Clinical Surgery, Tufts College Medical School. Surgeon-in-Chief, The Boston Dispensary. Visiting Surgeon, Cambridge Hospital, Cambridge, and the Boston Floating Hospital. Consulting Surgeon, Masonic Hospital, Shrewsbury and State Prison Colony Hospital, at Norfolk. His subject is "Torsion of the Pregnant Uterus. Report of Two Cases." Page 605. Address: 412 Beacon Street, Boston.

OTTENHEIMER, EDWARD J. B.S., M.D. University of Virginia Department of Medicine 1922. F.A.C.S. Chief, Surgical Service, Windham Community Memorial Hospital. His subject is "Postoperative Gas Pains." Page 608. Address: 29 North Street, Willimantic, Connecticut.

PETRONE, FRANCIS J. B.S., M.D. Harvard University Medical School 1926. Assistant Surgeon, Union Hospital, and General Hospital, Fall River, Mass. Address: 140 Winter Street, Fall River, Mass. Associated with him is

VIEIRA, EDWIN. A.B., M.D. Harvard University Medical School 1931. Externe, Medical Service O.P.D. Rhode Island Hospital, Providence, R. I., and Cardiac Service O.P.D. Rhode Island Hospital, Providence, R. I. Address: 221 Warren Avenue, East Providence, R. I. Their subject is "Incarcerated Bladder in a Scrotal Hernia. A Case Report." Page 614.

EDWARDS, EDWARD A. M.D. Tufts College Medical School 1928. Assistant in Surgery, and Member of the Circulatory Clinic, Boston City Hospital. Assistant in Surgery, Tufts College Medical School. His subject is "The Arteriographic Comparison of Thrombo-Angiitis Obliterans and Arteriosclerosis." Page 616. Address: 1571 Beacon Street, Brookline, Mass.

MOSCHCOWITZ, ELI. A.B., M.D. Columbia University College of Physicians and Surgeons 1900. Associate Physician, Mt. Sinai Hospital, New York City. Consulting Pathologist, Beth Israel Hospital, New York City. Consulting Physician, Beth-El Hospital, Brooklyn, N. Y. His subject is "Allergy to Life: An Interpretation of the Neurotic Constitution." Page 617. Address: 25 West 68th Street, New York City.

JOHNSON, C. K. M.D. University of Vermont College of Medicine 1899. F.A.A.P. Professor of Pediatrics, University of Vermont. Pediatrist, Mary Fletcher Hospital, Bishop DeGoesbriand Hospital, Fanny Allen Hospital, Elizabeth Lund Home and the Home for Destitute Children. His subject is "Atropine Fever in Early Infancy." Page 620. Address: 266 Main Street, Burlington, Vermont.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A STATED meeting of the Council of the Massachusetts Medical Society will be held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Wednesday, October 2, 1935 at 12 o'clock, noon.

Business:

1. Call to order at 12, noon.
2. Reading record of last meeting in abstract.
3. Report of Committee of Arrangements for Annual Meeting.
4. Report of Committee on Membership and Finance.
5. Reports of committees to consider petitions for restoration to the privileges of fellowship and appointment of new committees.
6. Appointment of an auditing committee.
7. Fill any vacancies in the offices of the Society.
8. *Incidental Business.*

ALEXANDER S. BEGG, *Secretary.*

Boston, September 25, 1935.

Councilors are asked to sign one of the two attendance books before the meeting.

SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. KICKHAM, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.
R. S. TITUS, M.D., *Secretary*, 472 Commonwealth Ave., Boston, Mass.

TRICHOMONAS VAGINALIS

Trichomonas vaginalis is a fairly common condition responsible for many cases of annoying leukorrhea. To make the diagnosis it is necessary only to place a cotton swab on an applicator, rub it firmly over the vaginal walls, and wash it off in a few cc. of warm normal saline solution. From this a hanging drop can be made, or a few drops may be placed under a cover slip on a slide. Examined under the high dry lens of the microscope the characteristic protozoa is readily identifiable, having moving flagella at one pole and several cilia at the other. It is pyriform or spindle shaped, larger than a pus cell but smaller than an epithelial cell.

In the presence of an acute vaginitis from this parasite the smear usually shows, in addition to the trichomonads, large numbers of pus cells, but as the condition is improved by treatment the pus cells diminish rapidly in number leaving the usual preponderance of normal epithelial cells. The discharge produced is often very profuse, yellowish-white, thin or mucoid, sometimes foamy, with a foul odor; it appears on a vaginal wall which is inflamed and frequently covered with fine petechiae. In addition to the discharge the patient usually complains of an itching or burning sensation at the introitus and of dyspareunia. Dysuria is often present; frequency is not so common.

It is trite to remark that the almost countless forms of treatment which have been recommended only suggest the many failures of each. Space does not permit even a résumé of the various measures which have been advocated. The writer has been so well satisfied, however, with a method which was suggested to him by Dr. Mortimer Hyams of New York that he has had no extensive experience with any other, and will here limit his discussion to this treatment.

The vagina is scrubbed with cotton saturated in half strength tincture of green soap and left for five minutes. The soap is next thoroughly washed out with an irrigation of hot mercury oxycyanide solution 1:3000. A speculum is introduced, the vaginal walls dried with cotton gauze, and two drams of one per cent picric acid in glycerine poured in. The vagina is then packed with a gauze bandage and the patient is directed to remove the packing twenty-four hours later. For the first two or three weeks the treatments should be given every second day, but after that they may be spaced farther and farther apart. In addition to these treatments the patient is advised to take a slow two quart douche of hot 1:3000 mercury oxycyanide solution each night before retiring except on those days when she has been treated. The douches are continued through the menstrual periods.

The patient should not be considered cured until she has been found free of any trichomonads when examined directly after the menstrual period for several consecutive months. And because recurrences are not uncommon it is safer to advise reëxamination at the end of another six months. It must be borne in mind, however, that there is at least a theoretical possibility of mercury poisoning if the douches are continued indefinitely, and for this reason their use should be limited to a few months at a time.

A high percentage of cures is certain with almost any of the recognized treatments if the patient is coöperative and the physician persistent in his efforts. And the woman who has had to wear perineal pads constantly or douche daily for months or years because of this foul discharge is most appreciative of the result.

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

MISCELLANY

THE SUCCESSOR TO DR. MAGRATH

With the announcement of the resignation of Dr. George Burgess Magrath from the position of medical examiner of Suffolk County because of ill health, His Excellency, Governor Curley, plans to advance Dr. William J. Brickley, associate examiner, to fill this position when Dr. Magrath vacates the office. The acceptance of Dr. Magrath's resignation will be deferred in order to allow him to qualify for a pension under the county retirement system. This is in recognition of the long and valuable service rendered by Dr. Magrath.

With the advancement of Dr. Brickley, the Governor plans to appoint Dr. Cornelius J. O'Leary to the position of Associate Medical Examiner, according to reports in the daily press.

Dr. Brickley has enjoyed the confidence and respect of the citizens of Suffolk County for the energy and ability displayed in his attention to the duties of the office of Associate Medical Examiner. By education and experience he is well fitted for the higher position.

Dr. O'Leary was born in 1902, graduated in medicine from the Tufts College Medical School in 1933 and served as interne at St. Elizabeth's Hospital. He is a member of the staffs of St. Elizabeth's, the Cambridge and Waltham Hospitals. He is bacteriologist for the Boston Department of Health and a Fellow of the Massachusetts Medical Society.

ANTERIOR POLIOMYELITIS CASES FOR 1935

WEEKLY LIST, SEPTEMBER 16-21

City or Town	
Attleboro	1
Fall River	7
North Attleboro	1
Westport	2
Braintree	1
Dedham	1
Medfield	1
Medway	1
Norwood	3
Quincy	8
Scituate	1
Walpole	2
Weymouth	2
Arlington	1
Boston	37
Brookline	1
Cambridge	5
Chelsea	2
Everett	1
Lexington	1
Malden	5
Maynard	2
Medford	1
Melrose	1
Newton	3
Revere	3
Somerville	7
Stoneham	1
Waltham	4
Wellesley	1
Amesbury	1
Dracut	1
Gloucester	1
Haverhill	1
Lowell	1
Lynn	1
Marblehead	1
Methuen	1
North Andover	1
Peabody	1
Reading	1
Swampscott	1
Wakefield	1
Wilmington	1
Milford	1
West Boylston	1
Worcester	2
Hardwick	2
Holyoke	1
Springfield	4
Total	132

CONNECTICUT CITIES ARE STUDYING NON-PROFIT HOSPITAL SERVICE

In an address before the Clinical Congress of the Connecticut State Medical Society, September 18, Mr. Van Dyk, executive director of the Associated Hospital Service of New York, reported that plans are being considered for non-profit hospital service in New Haven, Hartford, New Britain, Norwich, Derby, Norwalk, Bridgeport, Torrington, Meriden, and Danbury.

TUFTS COLLEGE MEDICAL SCHOOL NEWS ITEMS

Dr. Siegfried J. Thannhauser, formerly Medical Director of the University of Freiburg, has been appointed Associate Professor of Medicine.

Four scholarships of the value of $1,000 each, known as the Commonwealth Fund Scholarships, are awarded annually to properly qualified medical students. The recipients of these scholarships must agree to practice medicine in a rural community for a period of three years. The scholarships are awarded with the understanding that they will be renewed for each of the years required for graduation in medicine, provided that the record of the recipient warrants such extensions. Applicants must be residents of Massachusetts. The following selections have been made for the coming year:

Walter F. Crosby, Danvers, Mass., Bowdoin, '35.

Bronislaus A. Galuszka, Chicopee Falls, Mass., Tufts, '35.

Marshall A. Lamb, Rockland, Mass., Harvard, '34.

Horatio J. Young, Florence, Mass., Johns Hopkins, '35.

It is anticipated that the tuition of the medical school will be increased to $500.00 a year, and the enrollment limited to 100, beginning with the fall of 1936.

FROM NAVAL CUSTOMS, TRADITIONS AND USAGE BY LT. COMMANDER LELAND P. LOVETT, U. S. NAVY

UNITED STATES NAVAL INSTITUTE, ANNAPOLIS, MARYLAND, 1934, PAGES 232-233

Gun, Son of a. In the early days, sailors were permitted to keep their "wives" on board. The term was actually used to refer to children born alongside the guns of the broadsides. In fact, the expression questioned the legitimacy of anyone. The old definition of a man-o'-war's man was,

"Begotten in the galley and born under a gun. Every hair a rope yarn, every tooth a marline spike; every finger a fish hook and in his blood right good Stockholm tar."

A British officer commanding a brig off the Spanish coast in 1835 wrote in his diary,

"This day the surgeon informed me that a woman on board had been laboring in child for twelve hours, and if I could see my way to permit the firing of a broadside to leeward, nature would be assisted by the shock. I complied with the request and she was delivered of a fine male child."

The Gunnery Department surely made a perfect score.—*Submitted by Dr. John P. Monks.*

THE APPOINTMENT OF DR. JOHN A. HUFFMIRE

Dr. John A. Huffmire of Huntington, Massachusetts, has been appointed Associate Medical Examiner of the second Hampshire District and has taken the oath of office.

Dr. Huffmire is a graduate of the Middlesex College of Medicine and Surgery, Class of 1924.

THE APPOINTMENTS OF DR. JOHN V. GALLAGHER AND DR. IRVING F. ARMSTRONG

Governor Curley has appointed Dr. John V. Gallagher of Milford, medical examiner of the sixth Worcester District, to succeed the late Dr. George Curley, and Dr. Irving F. Armstrong of Hudson, medical examiner of the ninth Middlesex District, to succeed Dr. Norman M. Hunter.

MORTALITY RATES

Telegraphic returns from 86 cities, with a total population of thirty-seven millions for the week ending September 7, indicate a mortality rate of 9.4, as against a rate of 10.1 for the corresponding week of last year. The highest rate (17.8) appears for Nashville, Tenn., and the lowest (3.4) for Somerville, Mass. The highest infant mortality rate (15.5) appears for Cincinnati, Ohio, and the lowest for Bridgeport, Conn., Canton, Ohio, Duluth, Minn., Evansville, Ind., Jersey City, N. J., Long Beach, Calif., New Bedford, Mass., Schenectady, N. Y., Seattle, Wash., Somerville, Mass., South Bend, Ind., Spokane, Wash., Springfield, Mass., Tacoma, Wash., Utica, N. Y., and Yonkers, N. Y., which report no infant mortality.

The annual rate for 86 cities is 11.6 for the thirty-six weeks of 1935, as against a rate of 11.5 for the corresponding period of the previous year.—*Bureau of the Census.*

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1935

Disease	Aug., 1935	Aug., 1934	5 Yr. Average*
Anterior Poliomyelitis	483	16	142
Chickenpox	97	87	103
Diphtheria	23	51	109
Dog Bite	1117	1031	721
Epidemic Cerebrospinal Meningitis	4	2	6
German Measles	89	15	25
Gonorrhea	546	680	661
Lobar Pneumonia	123	90	83
Measles	130	95	199
Mumps	225	131	148
Scarlet Fever	175	179	249
Syphilis	444	401	351
Tuberculosis Pulmonary	324	340	321
Tuberculosis Other Forms	35	40	37
Typhoid Fever	15	24	34
Undulant Fever	2	0	
Whooping Cough	293	596	568

*Based on the figures for the preceding 5 years.

RARE DISEASES

Anterior poliomyelitis — 483 cases (see weekly lists).

Anthrax was reported from Sheffield, 1.

Dysentery (bacillary) was reported from Somerville, 1; Watertown, 1; total, 2.

Encephalitis lethargica was reported from New Bedford, 1; Swampscott, 1; total, 2.

Epidemic cerebrospinal meningitis was reported from Belchertown, 1; Cambridge, 1; Medford, 1; Norwood, 1; total, 4.

Malaria was reported from Watertown, 1.

Pellagra was reported from Boston, 1; Watertown, 1; total, 2.

Septic sore throat was reported from Athol, 1; Boston, 3; Brookline, 1; Lynn, 1; Winthrop, 1; total, 7.

Tetanus was reported from Fall River, 1; Hingham, 1; Watertown, 1; total, 3.

Trachoma was reported from Boston, 2; Worcester, 1; total, 3.

Trichinosis was reported from Boston, 1; Methuen, 1; total, 2.

Typhus fever was reported from Beverly, 1.

Undulant fever was reported from Cambridge, 1; Greenfield, 1; total, 2.

Infantile paralysis was at its highest reported August incidence with foci in Boston and Fall River and a scattering of cases throughout the eastern part of the state.

Diphtheria was at its lowest reported monthly incidence in the history of the state.

Lobar pneumonia continues to show an increase over last year's figures, which may mean an increased prevalence throughout the coming winter.

German measles had the usual summer decline after a year of epidemic incidence, with a possibility of another year of increased prevalence in 1936.

Typhoid fever, with a total of 76 reported cases to date, shows no increase over the 1934 record low figure.

Tuberculosis, pulmonary and other forms were reported to about the usual expectancy.

Epidemic cerebrospinal meningitis, measles, mumps, and chickenpox showed nothing remarkable.

Scarlet fever and whooping cough are running lower than in 1934.

THE CAMPAIGN TO DIMINISH AUTOMOBILE ACCIDENTS

The Boston Traveler is engaged in a campaign to reduce the number of automobile accidents and in order to secure the influential coöperation of doctors has deposited at the Boston Medical Library copies of the Safe Driving Pledge with the request that doctors be asked to sign and forward them.

This is a worthy movement and doctors may well participate in giving assurance of coöperation by signing the pledges under seven headings as follows:

1. To drive at moderate speed and on own side of road.
2. Not to pass cars on curves or hills.
3. To stop at stop signs.
4. Not to jump traffic lights.
5. In city traffic to be particularly watchful for pedestrians stepping into the line of traffic from parked cars.
6. Always to give hand signals showing my intention of turning to the left, to the right, or stopping, and not to leave the curb from a parked position without giving a signal.
7. To be fair to other drivers in all respects and to refrain from reckless driving.

CORRESPONDENCE

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

September 18, 1935.

Editor, *New England Journal of Medicine*,

I am writing you the following report to establish what I think is originality of action for the Middlesex South District Medical Society.

Recently a meeting of the Councilors of the Middlesex South District Medical Society was called by its President, Dr. Sumner H. Remick. The meeting was held at the Middlesex County Sanatorium. Thirty-four out of a total of fifty-one Councilors representing this District were present.

Many problems pertinent to the practice of medicine were discussed. Such problems as "the abuse of charity hospitals," "medical legislation," "postgraduate study," "relief for indigent physicians" and "program" were discussed and certain plans of procedure were evolved.

Dr. Charles E. Mongan, the President of the Massachusetts Medical Society, was present. He acquainted the Councilors with the medical economic situation and spoke about the future activity of the Society along these lines.

The meeting of the Council of the Middlesex South District Medical Society will no doubt, in the future, become an important part of the activity of this Society and will therefore place the Councilors in a better position to assume their responsibility toward the Massachusetts Medical Society.

Very truly yours,

ALEXANDER A. LEVI, M.D., *Secretary*.

RECENT DEATHS

MINDLIN—CARL MINDLIN, M.D., of 105 Emerson Street, Haverhill, died August 20, 1935. He was born in Russia in 1884 and early in life went to New York City where his early education was acquired. He graduated from the Long Island College of Medicine in 1910 and served as intern at the Long Island Hospital. He first settled in Manchester, New Hampshire, and soon afterward moved to Haverhill where he maintained an active practice for twenty-five years. He was a Fellow of the Massachusetts Medical Society and the American Medical Association. He is survived by his widow, Mrs. Ada Mindlin; a daughter, Barbara; a brother in Russia and a sister in New Jersey.

TRAINOR—JOHN BRETT TRAINOR, M.D., of 1517 South Main Street, Fall River, Massachusetts, died September 20, 1935, at St. Anne's Hospital.

Dr. Trainor was born in 1868 and graduated from McGill University Faculty of Medicine in 1897. He joined the Massachusetts Medical Society in 1901 soon after opening an office in Fall River.

He was a Fellow of the American Medical Association, and served on the staffs of the Union Hospital and St. Anne's Hospital of Fall River. He was especially devoted to x-ray work.

He was affiliated with the Knights of Columbus, the Ancient Order of Hibernians, the Royal Arcanums, and the Catholic Order of Foresters. Dr. Trainor is survived by his widow, Mrs. Margaret E. (Brady) Trainor, a sister, three brothers, and a nephew.

McQUAID—THOMAS BERNARD MCQUAID, M.D., of 42 Norwood Street, Everett, Massachusetts, died at

Webster, Massachusetts, September 19, 1935, while on vacation.

Dr. McQuaid was born in 1860, was a graduate of Holy Cross College, and received his medical degree from the Dartmouth Medical School in 1896. He had practiced in Everett for more than thirty years and had served as physician to the local Board of Health.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association.

Dr. McQuaid is survived by a sister, Miss Elizabeth McQuaid, and two brothers, Michael McQuaid of Webster and Owen McQuaid of California.

BRYANT — JOHN BRYANT, M.D., of 120 Sargent Road, Brookline, died September 19, 1935. Dr. Bryant was born in Cohasset, Massachusetts, in 1880, the son of the late John Bryant and Charlotte Olmsted Bryant. His premedical education was acquired at the Milton Academy and Harvard College. His medical degree was conferred by the Harvard University in 1907. Early in his medical career, he spent two years abroad in research work. On his return he served as assistant physician at the Robert Breck Brigham Hospital. He entered military service during the war with the title of captain and was advanced to the rank of major with assignment for duty at the surgeon-general's office in Washington.

After the war he was elected secretary to the American Gastro-Enterological Association and later became its president. Dr. Bryant was especially interested in the problems of convalescence and had written several articles on this subject and was consultant to the Burke Foundation for Convalescents in New York City.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association, and a member of the New England Pediatric Society, the Harvard Club, the Somerset Club, and the Society of Mayflower Descendants.

He is survived by his widow, Mrs. Adelaide Barnes Bryant, and two brothers, Edward Bryant of Bolton, New Hampshire, and Owen Bryant of Tucson, Arizona.

NOTICES

MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, October 3, in the amphitheater of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays, from October to May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian.

ERRATUM

The caption of the article by Milton S. Lloyd, M.D., published on page 101, issue of July 18, 1935, was incorrect. It should have read "The Clinical Classification and Early Diagnosis of Cancer of the Bronchus."

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, September 27, 12-1, Cheever amphitheatre.

Dr. William R. Morrison, Associate Professor of Surgery, will present:

1. Carcinoma of Distal End of Stomach. Billroth II Operation.
2. Carcinoma of the Transverse Colon with Partial Intestinal Obstruction. Resection with Intestinal Anastomosis.
3. Acute Appendicitis with Peritonitis.

Physicians and medical students are invited.

UNITED STATES CIVIL SERVICE EXAMINATION

Medical Supervisor (Psychiatric), $5,600 a Year
Department of the Interior

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than October 7, 1935.

Applicants must possess the following qualifications:

1. They must be citizens of the United States.
2. Education. — They must have been graduated from a recognized medical school with a degree of M.D.
3. Experience. — 1. They must have completed either a regular rotating or a psychiatric interneship of at least one year's duration, or must have had experience considered equivalent to such interneship.
   2. In addition, they must have had not less than five years of responsible and progressive specialized experience in neuropsychiatry, of which at least three years must have been in a senior administrative capacity in a mental hospital with not less than 300 daily average of patients under their care.
4. Age.—They must not have reached their fifty-third birthday on the date of the close of receipt of applications.

For more detailed information apply to the U. S. Civil Service Commission.

REPORT AND NOTICES OF MEETINGS

THE NEW HAMPSHIRE SURGICAL CLUB

The thirty-eighth annual meeting of the New Hampshire Surgical Club was held at the Farragut House, Rye Beach, New Hampshire, September 8, 1935.

Dr. Irving J. Walker, Clinical Professor of Sur-

gery, Harvard Medical School, read a paper on Infections of Clean Operation Wounds.

At the banquet, Dr. A. Warren Stearns, Dean of Tufts College Medical School, was the principal speaker.

A golf tournament was held on the Abenaqui links.

The following officers were elected for the ensuing year: Dr. Robert J. Graves of Concord, President; Dr. Richard W. Robinson of Laconia, Vice President, and Dr. Ralph E. Miller of Hanover, Secretary-Treasurer.

WORCESTER DISTRICT MEDICAL SOCIETY
1935-1936

Wednesday Evening, October 9, 1935
Rutland State Sanatorium, Rutland, Massachusetts.

PROGRAM

6:00 P.M. Dinner. (Complimentary by State Sanatorium.)

7:30 P.M. Scientific Program.

1. "X-ray Diagnosis Silicosis vs. Tuberculosis." Dr. David Zacks, Massachusetts Department of Public Health.

Discussion — Dr. W. Irving Clark, Worcester, Massachusetts.

2. "The Value of Blood Studies in the Selection of Cases for Thoracoplasty." Dr. Gulli Lindh Muller, Pathologist, Rutland State Sanatorium.

Dr. William Lynch, President; Dr. Erwin C. Miller, Secretary.

Wednesday Evening, November 13, 1935
Grafton State Hospital, North Grafton, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Evening, December 11, 1935
St. Vincent Hospital, Worcester, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Evening, January 8, 1936
Worcester City Hospital, Worcester, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Evening, February 12, 1936
Worcester State Hospital, Worcester, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Evening, March 11, 1936
Memorial Hospital, Worcester, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Evening, April 8, 1936
Hahnemann Hospital, Worcester, Massachusetts. Dinner and Scientific Program—Subjects of program to be announced later.

Wednesday Afternoon and Evening, May 13, 1936
Annual Meeting of Society—Time, place and details of program to be announced in an April issue of the *Journal*.

ERWIN C. MILLER, M.D., *Secretary*.

FAULKNER HOSPITAL CLINICAL MEETING

The first clinical meeting at the Faulkner Hospital for the season of 1935-1936 will be held on Thursday, October 3, 1935, at 5:00 P.M.

Subject: Several Cases of Unusual Vascular Lesions with Fatal Termination.

Discussion by Drs. Eugene E. O'Neil and John Homans.

All physicians are invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, SEPTEMBER 30, 1935

Wednesday, October 2—
†12 M. Clinico-Pathological Conference. Children's Hospital.

Thursday, October 3—
*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.
*5 P.M. Clinical Meeting—Faulkner Hospital.

Saturday, October 5—
*10-12. Staff rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

September 27—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 637.

September 27-28—New England Surgical Society. See page 549, issue of September 12.

September 30 - October 12—International Medical Post-Graduate Courses in Berlin. For further information apply to the Geschäftsstelle der Berliner Akademie für ärtzliche Fortbildung, Robert Koch—Platz 7 (Kaiserin Friedrich-Haus), Berlin N. W. 7.

October 3—Medical Clinic at the Peter Bent Brigham Hospital. See page 637.

October 3—Faulkner Hospital Clinical Meeting. See notice above.

October 6-20—Seventh Annual Training Course for Medical Reservists at the Mayo Clinic. See page 441, issue of August 29.

October 7-10—American Public Health Association will meet in Milwaukee, Wisconsin. For information address the American Public Health Association, 50 West 50th Street, New York City. See page 573.

October 14-19—Inter-State Postgraduate Medical Association of North America. See page 549, issue of September 12.

October 21 - November 2—1935 Graduate Fortnight of the New York Academy of Medicine. See page 898, issue of May 9.

October 28 - November 1—The Twenty-Fifth Clinical Congress of the American College of Surgeons. See page 1065, issue of May 30.

DISTRICT MEDICAL SOCIETY

WORCESTER DISTRICT MEDICAL SOCIETY

October 9 - May 13—Schedule of Meetings. See notice elsewhere on this page.